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"SOME ASPECTS OF INFANTILE PARALYSIS."[1]

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THE recent and severe epidemic of infantile paralysis in New Zealand (now fortunately abating) has caused much anxiety in the minds of many medical men. Just at the present time I feel they are not so much concerned with the clinical features of the disease. The points they would like to be elucidated are these: Are we likely to get a similar epidemic here in the near future? If so, what precautions if any, should or can be taken to guard against it Is serum treatment reliable? If so, is any attempt being made to procure or make it here? We are told that the infection is due to an ultra-microscopic organism (or virus) that can pass through a porcelain filter. How is it carried? Is human contact the only method of transmission? Or is the stable fly, the bed bug, or the flea responsible? These points will, I take it, be dealt with by Dr. R. Dick in his paper on the epidemiology of the disease.

While we are naturally anxious about the spread of the epidemic from New Zealand to Australia, it is well for us to remember infantile paralysis is endemic here and has been for many years. At the Children's Hospital cases are frequently cropping up. Dr. Ratcliff, the Medical Superintendent, has kindly supplied me with a list of patients admitted to the hospital from 1918 to 1924 inclusive, giving date, age, duration of illness, extent of lesion, type, time in hospital, result and district from which child came (see accompanying table).

In 1918 there were nine cases; in 1919 there was one case; in 1920 there were seventeen cases; in 1921 there were fifty-five cases; in 1922 there were seven cases; in 1923 there were forty cases; in 1924 there were forty-five cases. The total is one hundred and seventy-four cases. The deaths were eleven or 6.2%. Only two patients have been admitted with the disease during the first three months of 1925.

These figures indicate that there was a mild epidemic in 1921, 1923 and 1924. Most of the children were admitted in a fairly acute stage, the average time from onset of symptoms to admission in one hundred and forty cases being 6.5 days. Nine deaths took place within a few days. The type

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 7, 1925.

TABLE I.—SHOWING DETAILS OF PATIENTS ADMITTED TO THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN SINCE JANUARY, 1918, SUFFERING FROM ACUTE ANTERIO-POLIOMYELITIS.

Number	Sex	Age.		Date of Admission.	Duration of Illness, in Days.	Extent of Lesion.	Type.	Duration in Hospital, in Days.	Result.	District.
		Years.	Months.							
1	M	2	—	January 23, 1918	9	Left arm	Myelitic	14	Relieved	Randwick
2	M	10	—	February 16, 1918	7	Both legs	Myelitic	150	Relieved	Temora
3	M	—	11	February 21, 1918	7	Face	Encephalitic	75	Cured	Peakhurst
4	M	2	2	May 17, 1918	8	Both legs	Myelitic	2	Relieved	Cessnock
5	M	4	—	June 4, 1918	4	Both legs and back	Encephalitic	120	Relieved	Marrickville
6	M	6	—	June 26, 1918	10	Both legs	Myelitic	60	Relieved	Waterloo
7	M	5	—	July 5, 1918	150	Right arm	Myelitic	120	Relieved	Junee
8	F	2	4	July 7, 1918	42	Both arms, both legs	Myelitic	210	Relieved	Gunnedah
9	F	1	3	August 29, 1918	Not stated	Both arms, both legs	Myelitic	210	Relieved	Gunnedah
10	F	1	—	January 5, 1919	2	Right arm and leg, right side of face	Encephalitic	60	Relieved	Tennyson
11	M	2	—	January 20, 1920	4	Right arm	Myelitic	30	Relieved	Gordon
12	M	4	—	January 26, 1920	7	Both legs, arm, back	Myelitic	120	Died	Waverley
13	M	1	7	February 18, 1920	5	Left arm and leg, squint	Encephalitic	60	Relieved	Woollahra
14	M	1	3	April 2, 1920	5	Right leg	Myelitic	21	Relieved	Croydon
15	F	6	—	April 7, 1920	14	Both legs	Myelitic	60	Relieved	Naremburn
16	F	1	6	April 29, 1920	5	Right leg	Myelitic	30	Relieved	Randwick
17	M	1	6	May 7, 1920	8	Both legs	Myelitic	60	Relieved	Auburn
18	F	2	10	May 14, 1920	14	Both legs	Myelitic	120	Relieved	Arncliffe
19	F	2	—	May 15, 1920	150	Right leg	Myelitic	150	Relieved	Pitt Town
20	M	3	—	May 26, 1920	150	Both arms, legs, back and diaphragm	Myelitic	5	Died	Dunedoo
21	M	3	—	September 29, 1920	10	Both legs	Myelitic	7	Relieved	Pyrmont
22	F	2	—	October 5, 1920	21	Left arm, left leg	Myelitic	14	Relieved	Pyrmont
23	F	5	—	December 2, 1920	8	Both legs	Myelitic	21	Relieved	Paddington
24	F	—	6	December 15, 1920	4	Both arms, both legs, back and diaphragm	Myelitic	1	Died	Woollahra
25	M	3	6	December 15, 1920	14	Left side of face, left arm, left leg	Myelitic	15	Relieved	Waterfall
26	M	3	—	December 16, 1920	8	Both arms and legs	Myelitic	120	Relieved	Willoughby
27	M	3	6	December 29, 1920	5	Both legs, back, arms and diaphragm	Myelitic	2	Died	Paddington
28	M	6	—	January 1, 1921	7	Both legs and right arm	Myelitic	42	Relieved	Dulwich Hill
29	M	1	4	January 1, 1921	21	Right leg	Myelitic	7	Relieved	Redfern
30	F	2	8	January 2, 1921	7	Both legs	Myelitic	49	Relieved	Marrickville
31	F	4	—	January 5, 1921	2	Right leg and arm	Myelitic	30	Relieved	Darlinghurst
32	M	6	—	January 6, 1921	5	Both legs	Encephalitic	30	Relieved	Burwood
33	M	3	—	January 8, 1921	4	Face, right leg and arm	Myelitic	60	Relieved	Lidcombe
34	M	1	2	January 8, 1921	9	Legs, back	Myelitic	30	Relieved	?
35	F	1	2	January 10, 1921	6	Left leg	Myelitic	17	Relieved	Annandale
36	M	2	10	January 11, 1921	2	Left leg	Myelitic	42	Relieved	City
37	F	2	8	January 12, 1921	5	Right leg	Myelitic	60	Relieved	Collaroy
38	F	4	—	January 12, 1921	14	Right leg	Myelitic	14	Relieved	Hurstville
39	F	4	—	January 13, 1921	5	Both legs	Myelitic	14	Relieved	Moore Park
40	M	3	—	January 13, 1921	14	Left leg	Myelitic	60	Relieved	Woollongong
41	M	9	—	January 13, 1921	3	Both legs, both arms and diaphragm	Encephalitic	3	Died	Bankstown
42	M	3	—	January 14, 1921	21	Both legs	Myelitic	14	Relieved	Waterfall
43	M	3	—	January 16, 1921	42	Right arm and leg and (?) right side of face	Hemiplegic	14	Relieved	Pyrmont
44	M	1	8	January 17, 1921	7	Right arm, right leg, right side of face	Hemiplegic	30	Relieved	Drummoyne
45	F	4	—	January 18, 1921	4	Left leg	Myelitic	150	Relieved	Punchbowl
46	F	11	—	January 20, 1921	5	Both legs	Myelitic	240	Relieved	Edgecliff
47	M	4	—	January 21, 1921	7	Both legs	Myelitic	120	Relieved	Rockdale
48	F	1	6	January 24, 1921	14	Right leg	Myelitic	21	Relieved	Homebush
49	M	2	—	January 24, 1921	21	Right leg	Myelitic	2	Relieved	Newtown
50	M	1	2	January 25, 1921	4	Left side of face, (?) strabismus, right leg and arm	Encephalitic	60	Relieved	Zetland
51	M	3	—	January 29, 1921	7	Both arms, diaphragm	Myelitic	2	Died	Auburn
52	M	6	—	February 1, 1921	3	Both legs and left arm	Myelitic	60	Relieved	Punchbowl
53	F	5	—	February 2, 1921	21	Both legs	Myelitic	90	Relieved	Bexley
54	F	1	6	February 5, 1921	4	Right leg	Myelitic	13	Relieved	Waterloo
55	F	11	—	February 6, 1921	35	Both legs, palate, abdominal muscles	Cerebellar	90	Relieved	Botany
56	F	4	—	February 7, 1921	1	Right leg.	Abortive	10	Relieved	Pott's Point
57	M	8	—	February 8, 1921	2	Right leg.	Myelitic	8	Relieved	North Sydney
58	M	3	6	February 11, 1921	5	Left leg	Myelitic	35	Relieved	?
59	M	—	11	February 13, 1921	14	Both legs	Encephalitic	14	Relieved	Contracted at sea, s.s. *Boda*
60	F	3	—	February 14, 1921	4	Back, right leg	Myelitic	21	Relieved	Surry Hills
61	M	1	10	March 3, 1921	21	Right leg.	Myelitic	10	Relieved	Lidcombe
62	M	1	6	March 5, 1921	5	Back and neck, legs	Myelitic	60	Relieved	?
63	M	—	10	March 9, 1921	3	Right leg	Myelitic	14	Relieved	Mascot
64	M	3	—	March 9, 1921	35	Back, neck, abdominal muscles, both arms, both legs	Myelitic	270	Relieved	Flemington

TABLE I.—SHOWING DETAILS OF PATIENTS ADMITTED TO THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN SINCE JANUARY, 1918, SUFFERING FROM ACUTE ANTERIO-POLIOMYELITIS—*Continued*.

TABLE I.—SHOWING DETAILS OF PATIENTS ADMITTED TO THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN SINCE JANUARY, 1918, SUFFERING FROM ACUTE ANTERIO-POLIOMYELITIS—*Continued.*

Number	Sex	Age.		Date of Admission.	Duration of Illness, in Days.	Extent of Lesion.	Type.	Duration in Hospital, in Days.	Result.	District.
		Years.	Months.							
65	M	5	—	March 14, 1921	7	Left arm, back, right arm	Myelitic	42	Relieved	Glenbrook
66	M	2	2	March 15, 1921	8	Left leg and back, left arm	Hemiplegic	60	Relieved	Picton
67	F	6	—	March 16, 1921	4	Both legs	Myelitic	14	Relieved	Lidcombe
68	F	5	—	March 22, 1921	5	Right leg	Myelitic	21	Relieved	Miller's Point
69	F	5	—	March 24, 1921	4	Left arm	Myelitic	90	Relieved	Lidcombe
70	F	4	—	March 26, 1921	21	Both legs, right strabismus	Encephalitic	60	Relieved	Darlinghurst
71	F	8	—	March 28, 1921	7	Both arms and legs, back and diaphragm	Myelitic	210	Relieved	Campsie
72	F	1	8	March 30, 1921	7	Left arm	Myelitic	90	Relieved	Hawkesbury
73	F	2	6	April 17, 1921	42	Both legs and arms	Myelitic	90	Relieved	Narrandera
74	F	7	—	April 17, 1921	90	Both legs	Myelitic	210	Relieved	Byron Bay
75	M	4	—	April 19, 1921	4	Both legs, back	Myelitic	60	Relieved	Portland
76	M	—	6	April 29, 1921	63	Left arm	Myelitic	60	Relieved	Helensburg
77	F	2	—	May 4, 1921	6	Right leg	Myelitic	90	Relieved	Croydon
78	F	1	9	May 4, 1921	?	Right side of face	Myelitic	60	Relieved	Glebe
79	M	1	5	June 11, 1921	?	Right arm, both legs	Myelitic	210	Relieved	Alexandria
80	M	1	3	June 20, 1921	21	Right leg	Myelitic	60	Relieved	Bexley
81	F	6	—	July 9, 1921	90	Both legs	Myelitic	180	Relieved	Manly
82	F	4	—	December 23, 1921	7	Both legs	Myelitic	270	Relieved	Lizarow
83	M	4	—	January 6, 1922	7	Both arms, legs, back	Encephalitic	30	Relieved	Granville
84	F	2	—	March 7, 1922	21	Both legs	Encephalitic	1	Died	Auburn
85	M	4	—	March 20, 1922	4	Both arms	Myelitic	120	Relieved	Leichhardt
86	M	1	8	April 1, 1922	4	Left arm, left leg	Hemiplegic	35	Relieved	Kogarah
87	M	1	—	May 15, 1922	14	Both legs	Myelitic	90	Relieved	Redfern
88	M	9	—	June 1, 1922	12	Both arms and legs	Myelitic	11	Relieved	Grenfell
89	F	1	6	July 4, 1922	4	Right arm	Myelitic	7	Relieved	Leichhardt
90	M	1	6	January 6, 1923	2	Right arm	Myelitic	30	Relieved	Darlington
91	F	1	8	January 16, 1923	3	Both legs and right arm	Encephalitic	90	Relieved	City
92	M	1	2	January 24, 1923	4	Both legs	Myelitic	30	Relieved	Darlington
93	M	5	—	February 26, 1923	6	Both legs, back, arms	Encephalitic	60	Relieved	Petersham
94	F	5	—	March 4, 1923	3	Left leg	Myelitic	17	Relieved	Punchbowl
95	M	1	6	March 5, 1923	10	Both legs	Myelitic	42	Relieved	Forest Lodge
96	M	1	10	March 8, 1923	8	Left leg	Myelitic	35	Relieved	Rozelle
97	F	1	8	March 19, 1923	5	Right leg	Myelitic	9	Relieved	Darlinghurst
98	M	5	—	March 19, 1923	10	Both legs	Myelitic	42	Relieved	Lithgow
99	M	1	8	March 20, 1923	2	Eye Muscles, both legs and back	Encephalitic	60	Relieved	Redfern
100	F	2	10	March 27, 1923	18	Right leg	Myelitic	30	Relieved	East Sydney
101	M	2	6	April 3, 1923	?	Neck, back, both legs	Encephalitic	17	Relieved	Surry Hills
102	M	5	—	April 10, 1923	5	Both legs	Myelitic	210	Relieved	Glebe
103	M	6	—	April 11, 1923	4	Right leg, back	Myelitic	30	Relieved	City
104	F	1	5	April 11, 1923	7	Back and right leg	Myelitic	60	Relieved	Strathfield
105	F	2	—	April 19, 1923	14	Right leg	Myelitic	11	Relieved	Campsie
106	M	2	—	April 19, 1923	7	Both legs	Myelitic	14	Relieved	Balmain
107	F	1	8	April 26, 1923	11	Right leg	Myelitic	63	Relieved	Newcastle
108	F	3	—	April 28, 1923	5	Both legs, eye	Encephalitic	14	Relieved	Paddington
109	M	2	—	April 30, 1923	3	Right leg	Myelitic	14	Relieved	Pyrmont
110	M	1	2	April 30, 1923	10	Right leg	Myelitic	30	Relieved	Alexandria
111	F	2	6	May 3, 1923	6	Legs, abdomen, diaphragm	Myelitic	1	Died	Marrickville
112	M	—	7	May 3, 1923	14	Right arm and leg	Myelitic	10	Relieved	Newtown
113	M	3	—	May 3, 1923	14	Back, both legs	Myelitic	60	Relieved	Auburn
114	M	2	—	May 7, 1923	7	Right leg and back	Myelitic	35	Relieved	City
115	M	1	10	May 14, 1923	6	Left arm, right leg	Myelitic	30	Relieved	Petersham
116	F	4	—	May 16, 1923	14	Right arm, both legs	Myelitic	63	Relieved	Martin's Creek
117	F	1	—	May 17, 1923	7	Both legs and back	Myelitic	21	Relieved	Waterloo
118	M	1	—	May 18, 1923	30	Both legs, arms, back and eye	Encephalitic	210	Relieved	Lidcombe
119	F	2	—	May 22, 1923	7	Both legs	Myelitic	30	Relieved	Leichhardt
120	F	1	4	May 23, 1923	6	Right leg	Myelitic	21	Relieved	Bondi
121	F	6	—	May 24, 1923	14	Both legs, back	Myelitic	240	Relieved	Minto
122	F	2	—	May 28, 1923	1	Back	Myelitic	10	Relieved	Annandale
123	F	4	—	June 14, 1923	14	Both legs	Myelitic	12	Relieved	Pyrmont
124	M	1	5	June 19, 1923	6	Left leg	Myelitic	42	Relieved	Putney
125	M	5	—	June 20, 1923	6	Both arms, legs and back	Myelitic	180	Relieved	St. Peters
126	M	2	8	June 25, 1923	?	Left arm	Myelitic	7	Relieved	Parramatta
127	M	4	—	July 4, 1923	14	Both arms, both legs, back, right side of face, respiratory muscles	Myelitic	1	Died	Revesby
128	F	13	—	October 27, 1923	90	Right leg	Myelitic	30	Relieved	Yeoville, *viâ* Wellington
129	M	6	—	November 23, 1923	3	Diaphragm and abdominal	Encephalitic	1	Died	Ourimbah

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Number	Sex	Age. Years.	Age. Months.	Date of Admission.	Duration of Illness, in Days.	Extent of Lesion.	Type.	Duration in Hospital, in Days.	Result.	District.
130	F	2		January 5, 1924	6	Both legs	Myelitic	30	Relieved	Mascot
131	M	2		January 7, 1924	12	Left leg	Myelitic	12	Relieved	Bexley
132	M	4		January 8, 1924	7	Right leg	Myelitic	42	Relieved	Guildford
133	M	1	9	January 19, 1924	42	Left leg	Myelitic	60	Relieved	Cowra
134	M	2		January 21, 1924	60	Both legs left leg, back	Myelitic	60	Relieved	Granville
135	F	1	5	January 21, 1924	14	Right leg, back	Myelitic	150	Relieved	Woollahra
136	M	2		February 6, 1924	15	Both legs, back, arms	Encephalitic	42	Relieved	Gladesville
137	M	2		February 15, 1924	4	Both legs, back	Myelitic	21	Relieved	Balmain
138	M	2		February 15, 1924	5	Right arm	Myelitic	30	Relieved	Redfern
139	M	2		February 24, 1924	4	Left leg	Myelitic	90	Relieved	Guildford
140	F	2		February 29, 1923	2	Both legs	Myelitic	30	Relieved	Redfern
141	F	3	7	March 6, 1924	21	Both arms, legs, back, diaphragm	Myelitic	120	Died	Dungog
142	M	3		March 23, 1924	7	Both legs, back, both arms	Encephalitic	150	Relieved	Heathcote
143	M	2		March 8, 1924	1	Right leg	Myelitic	42	Relieved	Paddington
144	M	1	5	March 19, 1924	2	Left leg	Myelitic	35	Relieved	Drummoyne
145	F	3		March 13, 1924	2	Right leg	Myelitic	42	Relieved	Newtown
146	F	1	6	March 13, 1924	6	Left leg	Myelitic	49	Relieved	Drummoyne
147	F	1		March 19, 1924	2	Both legs, back	Myelitic	49	Relieved	Milson's Point
148	F	3		March 19, 1924	2	Both legs	Myelitic	21	Relieved	Alexandria
149	M	3		March 20, 1924	7	Both legs	Myelitic	90	Relieved	Surry Hills
150	F	2		April 2, 1924	14	Right leg	Myelitic	4	Relieved	Paddington
151	M	4		April 3, 1924	9	Both legs	Myelitic	150	Relieved	Wingham
152	M	2		April 5, 1924	5	Right leg	Myelitic	120	Relieved	Granville
153	M	8		April 6, 1924	90	Both legs	Myelitic	180	Relieved	Coonabarabran
154	F	4		April 8, 1924	5	Both legs	Myelitic	120	Relieved	Leichhardt
155	M	6		April 10, 1924	14	Left arm and left leg	Hemiplegic	14	Relieved	Paddington
156	F	2		April 22, 1924	7	Both legs and back	Myelitic	60	Relieved	Gladesville
157	F	2		April 23, 1924	60	Both legs	Myelitic	21	Relieved	Five Dock
158	M	1	7	April 30, 1924	7		Myelitic	90	Relieved	Gosford Race-course
159	M	3		April 30, 1924	14	Both legs, back	Myelitic	30	Relieved	Gosford Race-course
160	M	2		May 2, 1924	4	Right leg	Myelitic	42	Relieved	Artarmon
161	F	2		May 14, 1924	7	Left leg	Myelitic	42	Relieved	Five Dock
162	F	4		May 17, 1924	14	Both legs	Myelitic	42	Relieved	Arncliffe
163	F		10	May 20, 1924	6	Right arm	Myelitic	56	Relieved	Mortlake
164	M	2		May 24, 1924	4	Right arm and leg	Myelitic	90	Relieved	Camperdown
165	M	3		May 28, 1924	150	Right arm, both legs, back	Myelitic	180	Relieved	Geurie
166	F	1	9	June 2, 1924	7	Both arms, left leg	Myelitic	49	Relieved	Gladesville
167	M	1		June 15, 1924	42	Both legs	Myelitic	60	Relieved	Campsie
168	F	1	5	June 17, 1924	21	Right leg	Myelitic	21	Relieved	Toongabbie
169	M	2		June 30, 1924	7	Both legs, back	Myelitic	60	Relieved	St. Peters
170	M	2		July 1, 1924	14	Both legs, both arms, back	Myelitic	120	Relieved	Darlinghurst
171	F	11		July 14, 1924	28	Left arm, left leg	Myelitic	60	Relieved	Yarrabandi, near Parkes
172	M	4		August 5, 1924	10	Left leg	Myelitic	90	Relieved	Campsie
173	M	1		September 27, 1924	7	Left leg	Myelitic	30	Relieved	Campsie
174	F	2		November 3, 1924	120	Right leg	Myelitic	21	Relieved	Bulli

was myelitic in 86% of the cases and encephalitic in 10%. The children came from all parts of the State, one hundred and thirty from the City and suburbs and forty-four from the country.

General Symptoms.

Batten[1] describes the symptoms as follows:

> The disease usually begins with a sudden onset; there is commonly fever of moderate degree, which lasts for two or three days. The child is often drowsy, complains of headache and pains in limbs, which may be so acute as to give rise to the impression that the child has rheumatism. The pain is greatly accentuated by any movement. . . . In some few cases vomiting and diarrhœa may occur. The onset is sometimes attended with convulsions, and such convulsions may occur in cases which at a later stage show no sign of cerebral involvement.
>
> The neck may be stiff and even retracted and any movement forward causes pain. In the drowsy condition the child may pass urine and fæces involuntarily, but even when no such drowsiness exists loss of control over the sphincters or retention of urine may be present and last for days. . . . Another type of onset must be recognized in which a child is put to bed apparently in perfect health, and on the following morning a flaccid paralysis of one leg noticed. There is no pain on passive movement and there is apparently no constitutional disturbance.

Draper[2] under the heading of symptomatology says:

TABLE II.

SHOWING SUMMARY OF CASES DESCRIBED IN TABLE I.

Year.	Admissions.		Discharged.				Died.		Total.
			Cured.		Relieved.				
	M.	F.	M.	F.	M.	F.	M.	F.	
1918	7	2	1	0	6	2	0	0	9
1919	0	1	0	0	0	1	0	0	1
1920	10	7	0	0	7	6	3	1	17
1921	29	26	0	0	27	26	2	0	55
1922	5	2	0	0	5	1	0	1	7
1923	23	17	0	0	21	16	2	1	40
1924	27	18	0	0	27	17	0	1	45
Total	101	73	1	0	93	69	7	4	174

Death rate = 6.8%.

Acute poliomyelitis is not essentially a disease of the nervous system; it is a general systemic infection, in the latter part of whose course paralysis is an accidental and incidental occurrence and fortunately a comparatively infrequent one. With the development of the specific serum therapy, it is vital that we should recognize the disease in the absence of the paralysis, or before muscular weakness appears in those cases whose destiny may be paralysis. But equally for the purpose of limiting epidemics every effort should be made to diagnose the infection early. Obviously from what has been said there are two distinct phases in the course of the malady when it goes on to paralysis. The first a systemic reaction to a generalized infection, the second a particular highly specialized reaction of the meninges, brain and cord, to a specific toxin. These two phases of the disease are quite distinct and bear . . . certain relationships to one another. Thus either phase may be so light as to escape detection, nor does the intensity of one necessarily bear any relation to the violence of the other. The two symptom groups may be sharply separated by an interval of well-being, which may last from four hours to eight days, or the systemic period may merge imperceptibly with that of the meningeal invasion. The first phase frequently (50% to 80%) occurs alone and it is this form which has been called "abortive."

Diagnosis.

Everybody knows that a diagnosis is rarely made in the first phase of the disease. Holt[3] says:

> The recognition of acute poliomyelitis before the occurrence of paralysis is impossible, except by lumbar puncture.

In private practice we all know that lumbar puncture is futile unless we have the services of a pathologist. The added objections to doing it in a private house are according to Frauenthal[4] sudden death, infection, the fact that friends of the patient may attribute the paralysis to puncture.

In hospital lumbar puncture is carried out in all such cases, but even with all the resources of a well equipped hospital it is not always possible to make a diagnosis. Holt[3] says that the usual fluid found in acute poliomyelitis resembles that of tuberculous meningitis, which in some cases is closely simulated in clinical symptoms.

In time and as we advance in knowledge no doubt many of these conditions will be recognized in the first stage, at the present time we must acknowledge that (except in the presence of an epidemic) poliomyelitis is rarely diagnosed before paralysis has taken place. How then can a general practitioner (or anybody else) be expected to recognize a sporadic case occurring in his practice? The doctor may be called to see a child taken suddenly ill, perhaps it has vomited and has had loose motions. He finds it with a raised temperature, it is cross, irritable and difficult to examine. Nothing definite is made out. It may be sickening for almost anything. This may be some gastro-intestinal upset due to improper food. Why should he diagnose infantile paralysis? To suggest such a thing in the absence of an epidemic would put him down as an alarmist. And yet when the child after a few days' illness is found to have lost power in one or more limbs, he is often blamed for not having discovered it before and he is accused of "wrongly treating" the child. More especially is this likely to happen if he has been induced to label the condition that he has been treating.

If Draper[2] is correct when he says that from 50% to 80% of the cases abort and do not go on to paralysis, we can readily understand why the disease spreads and how difficult it must be to control because perhaps not half the cases are notified.

There are also certain milder forms of poliomyelitis that escape notification at the time. A child may have been very slightly ill for a very short time, not sufficiently sick for the people to call in a doctor. After a time it is noticed that the child is slightly lame or has a weakness in one arm. On examination it is found that there is some slight wasting of the limb and paresis of one or more muscles. When this is discovered the condition has to be explained to the parents, in order to insure rest and prevent the weakened muscles from being over taxed. But the blow of hearing that the child has infantile paralysis is much softened when you are able to assure them that by this mild attack the child has in all probability acquired immunity.

Prognosis.

The prognosis as to life varies in different epidemics from 10% to 25%. Lovett[5] tells us:

> The prognosis as to function is a very practical matter and one on which the surgeon will be closely cross-examined by the parents, and in the present state of our knowledge the man of experience will in the early days of the disease express himself with great reserve in this matter. In the acute stage it is particularly unwise to express a definite opinion as to the final function. On the whole a severe onset seems more unfavourable than a mild onset, but in individual cases a severe onset may be followed by complete recovery, or a mild paralysis, and a mild attack by severe and extensive paralysis.

So we must always be very careful and guarded when questioned by relatives and it is well not to be too dogmatic when asked if the paralysis is likely to spread. As a rule the worst is known at once, but you must remember that occasionally the paralysis does spread. In nearly all the cases a certain amount of spontaneous improvement will take place, as the tenderness is going.

We have but few statistics as to complete recovery. The Massachusetts State Board of Health dealing only with two hundred and twelve cases in 1911 reported that fifty-seven patients (27%) had wholly recovered. We do know, as I said before, that the percentage of deaths and complete recoveries varies with each epidemic. We also know that there are always far more patients left more or less permanently crippled than those that completely recover.

Treatment.

Serum Treatment.

Serum treatment may be instituted, firstly with serum obtained from the blood of those who have recovered from the disease and secondly with immune horse serum as prepared by Rosenow.[6]

There seems to be ample evidence of the beneficial effects of the administration of serum obtained from recovered persons, but in this form it can only have a very limited field of usefulness because of the difficulty of getting it.

Rosenow's serum is prepared by repeated injections of increasing doses of freshly isolated strains of the pleomorphic streptococcus. He says it has a curative power in poliomyelitis, especially when given in the early stage.

Draper says that a case for the anti-poliomyelitis serum has still to be made out convincingly. It seems to me that plenty of time has elapsed now for evidence to be obtained as to the efficacy of this serum. If it has been proved that this serum lowers the death rate and lessens the paralysis, then it is the obvious duty of the Federal Health authorities to obtain a supply or to set about manufacturing here in Australia at once. Why wait until the epidemic is upon us? The treatment by drugs is futile.

General Treatment, Massage and Splints.

In the acute stage then in the absence of a serum the less you do the better. Let the child lie in bed in the position it finds most comfortable. Give plenty of water. Splinting in the very early stages may greatly aggravate the child's sufferings.

Frauenthal[4] says:

> Do not make the mistake of applying any method of immobilization to these tender anguished frames. The inquisition furnished no more cruel measure than the application, which has been advocated of a plaster jacket during the acute stage of poliomyelitis.

And Lovett[5] says:

> In the early stage rest in bed must be strictly enforced in all cases. In the moderate and severe grades the patients are too ill to be elsewhere than in bed, but in the slighter cases it is well to remember that the pathological condition is a hæmorrhagic myelitis, and that the function of the affected members means function of their motor nerve cells and that on general principles such function is probably harmful.
>
> In all grades of the affection it is obviously indicated to secure the maximum degree of rest and quiet even where it is not necessitated by general prostration, pain and tenderness. During this stage the joints will not become anchylosed, muscles will not become hopelessly wasted, the circulation of the affected members will not become seriously upset if the patient is let alone, whereas if the patient is not let alone, but is massaged, manipulated and treated with electricity *et cetera* during this stage, there is a strong probability that the recovery will be less extensive than it would otherwise have been. That massage during convalescence is harmful so long as the patient is tender, has been shown many times and the tenderness may evidently be prolonged for months by constant manipulation and massage.

I know quite well that the policy of "do nothing" does not appeal to the public or to some medical men. But it is our duty to educate in this as in many other matters.

Later on when the acute stage is passing off, to prevent contractions and over-stretchings splints of some kind must be applied. For the foot, leg and thigh nothing is more comfortable than a properly made plaster of Paris mould. In some cases quite in the early stage a plaster splint seems to give relief when applied to the lower limb and it enables the child to be moved without pain.

In from six weeks to two months or more (the time varying in each individual case), but as the tenderness is passing off, gentle massage and muscle training may be started. It must always be discontinued if it causes pain. No heavy massage is ever required. In the olden days when nothing was known about muscle education the routine treatment for infantile paralysis almost as soon as it was diagnosed, was massage, heavy massage, as often as possible. Those few people who could afford it sometimes engaged a masseuse and kept her in the house. Hardly any of the children so treated made any improvement while under this treatment. At the massage department of the Children's Hospital we have at the present time about one hundred children attending, all suffering in various ways and degrees from the damaging effects of infantile paralysis. About fifty attend three times a week and receive on an average about fifteen minutes' muscle education. The others come at periods of three to six months to have their boots and splints attended to.

One cannot help feeling sorry for many of these poor women who are compelled to drag their children to a massage department three times a week in order that the child may get fifteen minutes' muscle education. For those living at any distance it means the loss of a whole afternoon and with many it must entail not only inconvenience, but much suffering. These children should have muscle training every day and twice a day. It is obvious that the only way the majority of them can get such treatment is from the parents. The mothers must be taught. With an intelligent woman there is no difficulty about this and when the importance is impressed upon her, she is quite eager and anxious to do anything to help her child. Only a small percentage of women are quite hopeless. When the mother has once been taught she need not bring her child more than once a week or less often.

Surgical Treatment.

Great advances have been made in this branch of surgery in the last ten years. But the surgeon must stay his hand and not be too eager to rush in until it is certain that no further improvement will take place and that the area of persistent paralysis can be definitely determined.

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(2) Draper: Nelson's "Loose Leaf Living Medicine," Volume II.
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(4) Frauenthal and Manning: "Infantile Paralysis," 1914.
(5) Lovett: "Treatment of Infantile Paralysis," Second Edition.
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THE PATHOLOGICAL ASPECT OF INFANTILE PARALYSIS.[1]

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It is right that I should preface my contribution with the statement that I have been requested to take part in this discussion at short notice and that my interests have not led me into the special domains of neurology and, therefore, I speak with some reluctance. Excepting only the examination of cerebro-spinal fluid, I have not done any work on infantile paralysis. The Medical Secretary has, however, informed me that this meeting has been arranged somewhat hurriedly, for it was thought advisable to bring this disease specially to the notice of medical practitioners, particularly those in general practice, because of the existence of an epidemic in New Zealand during the present summer.

Historical.

The modern history of infantile paralysis is briefly as follows. During the nineteenth century it was carefully studied by Heine in Germany and by Medin in Sweden and the latter recognized various clinical types, predominant being the spinal type, but also cerebral, bulbar, ataxic and polyneuritic. In 1905-1906 Wickmann, also of Sweden, described a grave type resembling Landry's paralysis and by his recognition of the frequency of abortive cases made it possible to understand better the transmission of the disease from place to place.

In 1908 a new era began with the experimental study of the disease. Landsteiner and Popper and later Flexner and Lewis infected monkeys and it was soon demonstrated that the disease could be carried on from monkey to monkey and that the contagium was a filtrable virus. It was cultivated in artificial media in 1913 by Flexner and Noguchi and Koch's postulates were satisfied by successful re-inoculation of monkeys.

To proceed in point of time, though deviating somewhat from the Heine-Medin disease, we have in 1917 the recognition by von Economo in Austria and in 1918 by Hall and Harris in England of the disease called *encephalitis lethargica* or epidemic encephalitis. It is worth remembering that in England at any rate, this disease preceded the epidemic of influenza in 1918 and that this epidemic form of encephalitis is possibly due to a filtrable virus allied to, but certainly not identical with, that of infantile paralysis.

It is interesting to take the 1906 edition of Osler's "Practice of Medicine" and the 1920 edition of Osler and McCrae's book and to compare the classification of the acute inflammations of grey matter, of brain and spinal cord, not due to trauma or intoxications or to the bacteria or viruses of the known acute infections.

1907.	1922.
1. Acute anterior polio myelitis.	1. Acute poliomyelitis.
2. Acute and subacute polio-myelitis of adults.	2. Epidemic or lethargic encephalitis.
	3. One type of cerebral palsies in children (the acute sporadic encephalitis of children with consecutive hemiplegia).
	4. Acute encephalo-myelitis, corresponding in histological details with polio-myelitis.

The chief progress is seen to be in the recognition of the cerebral localizations of the damage produced by these types of virus and by their differentiation mainly on clinical and histological criteria from each other and from the associated encephalitis occurring in the acute specific fevers and in microbic meningitis.

The Lesions in Infantile Paralysis.

To quote Osler and McCrae:

> We can no longer regard it as an affection limited to the anterior horns of the gray matter of the spinal cord, but a widespread polio-myelo-encephalo-meningitis. . . . Swelling of the spleen and a marked general hyperplasia of the lymphoid apparatus have been found. . . . The *pia mater* is hyperæmic and moist, but without exudate. Cases in which the cerebral symptoms have been pronounced, show swelling and flattening of the convolutions, with hyperæmia of the gray matter and here and there small hæmorrhages. The changes in the spinal cord are very characteristic. The meninges are moist, the pia is hyperæmic, sometimes with small capillary hæmorrhages. On section the anterior surface bulges, the gray matter is hyperæmic, appearing as a reddened H, or the redness is limited to the anterior horns, which may show spots of hæmorrhage. . . . Microscopically there is small-celled infiltration about the vessels of the meninges, most marked in the lumbar and cervical swellings. The infiltration extends into the fissures of the cord and follows the blood vessels. . . In the cord itself the smaller blood vessels are distended, hæmorrhages occur in the gray matter, there is marked perivascular infiltration, chiefly of lymphocytes, which collect about the vessels, forming definite foci. Sometimes the majority of the cells are polynuclear leucocytes. The ganglion cells, usually those of the anterior horns, degenerate and gradually disappear, changes probably secondary to the acute vascular alterations. . . . In the fatal cases there are changes in the medulla and pons of much the same nature, but the ganglion cells rarely show such widespread destruction.

In some epidemics cases of acute bulbar paralysis are apparently referable to the same virus.

Degenerate nerve cells may undergo phagocytosis and myelin sheaths and axis cylinders may be

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 7, 1925.

FIGURE I.
Spinal Cord in Acute Poliomyelitis, Showing the Swollen Ghost-Like Cells of Clarke's Column and the Leucocytic Infiltration.
(From Osler's "Modern Medicine.")

disintegrated in a similar way. Some nerve cells show only degrees of chromatolysis and as Muir points out:

> It is remarkable how little altered some of them are, even when surrounded by inflammatory changes, and the appearances suggest that the nerve cells suffer damage more from interference with their blood supply than from toxic action.

Wallerian degeneration in the axons follows death of the anterior cornual cells and the degenerated axons are replaced by interstitial tissue. When the acute inflammation in the cord subsides many of the damaged cells may recover with subsequent diminution of paralytic phenomena. The dead material in the cornua is absorbed and replaced by proliferation of neuroglia cells with subsequent sclerosis, leading sometimes to a naked-eye shrinkage of the cornua affected. It is believed that the anterior cornua are affected not so much by any selective action of the virus, as by the more abundant blood supply to this portion of the cord.

The Filtrable Virus of Poliomyelitis.

Monkeys are susceptible and the disease, though most easily produced by intra-cerebral inoculation, was first produced by intra-peritoneal inoculation of emulsions of infected nervous tissue. Thereafter successive monkeys may be infected by "passage." The virus will pass through porcelain filters and will maintain its virulence in glycerine for months, thus resembling the virus of rabies and vaccinia. It will resist freezing for long periods, its virulence diminishes at 45° C. and is killed within half an hour by a temperature of 50° C.. It resists desiccation and will live for some time in sterile water or milk. It resists a 1% solution of phenol for at least five days. It was cultivated by Flexner and Noguchi in 1913 in sterile ascitic fluid containing a portion of sterile rabbit kidney in which a portion of infected brain was inserted and the whole covered by paraffin. The fluid became opalescent on the fifth day and minute globoid bodies about 0.2 microns in diameter were found in pairs, chains and groups. Subcultures into similar media reproduced the disease in monkeys and the globoid bodies have been recognized apparently in sections of the brain of both patients and infected monkeys.

The disease has also been transmitted by subdural injection, by injection into the sciatic nerve, by rubbing the virus into the scarified mucous membrane of the nose, by subcutaneous injection, but, strange to say, enormous doses are necessary by the intravenous route. The virus both in the natural and experimental disease is concentrated in the central nervous system, but it has been found also in the abdominal sympathetic ganglia, in the lymph glands, in the tonsils and in the nasal mucosa, but is usually absent from the solid viscera, the blood and the cerebro-spinal fluid. Monkeys are the only animals that have been infected with certainty, though Rosenow claims to have infected rabbits.

The orthodox view is that the infection of the nervous system takes places usually from the nasal mucosa *viâ* the lymphatics of the olfactory nerves. In monkeys infected by the nasal route the olfactory lobe has been found infected prior to the rest of the brain. The enormous dose required to produce the disease by intravenous injections makes it unlikely that under natural conditions the virus is carried to the nervous system by the blood vessels.

Quoting Muir and Ritchie:

> There is evidence from experimental intravenous injections that the choroid plexus, which is the source of the cerebro-spinal fluid, prevents the passage of virus into the subdural space. It is likewise possible that in natural infection the virus may pass into the mesenteric nodes and thence be absorbed by the lymphatics of the spinal nerves.

There is no sufficient evidence of any intermediate agent of infection other than man himself and human carriers have been identified. It seems unlikely that the bites of a blood-sucking fly, such

FIGURE II.
Brain (Basal Nuclei) in Epidemic Encephalitis, Showing the Perivascular "Collars" of Small Round Cells.
(After McIntosh and Turnbull.)

TABLE SHOWING RESULTS OF EXAMINATION OF CEREBRO-SPINAL FLUID IN SEVERAL CONDITIONS.

Observation.	Encephalitis Lethargica.		Poliomyelitis.		Tuberculous Meningitis.	Brain Tumour.	Syphilis.
	Acute Stage.	Chronic Stage.	Initial Stage.	Paralytic Stage.			
Protein content	Inconstant, slight increase	Normal	Moderate increase	Moderate increase, sometimes excessive	Increased, sometimes excessive	Normal	Slight increase
Cell count	Usually increased, average 30	Normal	Increased, usually under 100	Increased, average 150	Increased, usually over 100	Normal except where meninges affected	Increased
Predominant cells	Lymphocytes	Lymphocytes	Polynuclear cells, 60%	Lymphocytes, but polynuclear cells present	Lymphocytes	Lymphocytes	Lymphocytes
Coagulum	Nil	Nil	Doubtful	Usually present	Usually present	Nil	Nil
Wassermann test	No reaction	No reaction	No reaction	No reaction	No reaction	No reaction	Positive

as *Stomoxys calcitrans*, play any part, as claimed by Rosenow, in the natural disease, because the blood is non-infective in small doses. It is interesting to note here that Rosenow claims to have found a pleo-morphic streptococcus in the brains of both patients and infected monkeys and that with this organism the disease can be reproduced. These views have not been generally accepted.

I will leave to Dr. Dick the discussion of carriers, of abortive attacks of the disease and of the epidemiology in general.

Immunity.

Muir and Ritchie state that no record of a second attack of poliomyelitis exists and monkeys successfully infected are afterwards immune. The serum of both man and monkey after recovery from the disease contain antibodies which will neutralize the virus *in vitro* and there is also a slight protective effect *in vivo* if injected intrathecally previous to massive intravenous inoculation of the virus. The serum of convalescents, if administered early, has some therapeutic effect in the way of arresting paralyses. From thirty-five to one hundred and twenty cubic centimetres have been given by both intrathecal and intravenous routes.

Laboratory Methods as Aids to Diagnosis.

The laboratory aids to diagnosis may be summarized as follows:

The isolation and recognition of the virus is not practicable as a routine method.

There is usually a leucocytosis, but this is apparently not very helpful in differentiating from meningitis and so forth in children.

In convalescence the serum of the patient neutralizes the virus, but this procedure can seldom be used in diagnosis.

The precipitin reaction of Rosenow cannot be generally accepted unless the organism isolated by him is accepted as specific.

There remains the careful examination of the cerebro-spinal fluid as the most useful and often essential procedure. The accompanying table shows the most useful criteria. I have followed J. R. Perdrau's findings in *encephalitis lethargica* and his summary of Durand's findings in acute poliomyelitis. It is interesting to note that in both diseases there is a biphasic reaction as a rule. Cerebro-spinal fever, pneumococcal and influenzal meningitis and meningitis due to pyogenic cocci, usually an extension from neighbouring foci, may be differentiated usually by the turbidity of the fluid, the enormous increase in cells, mostly polynuclear leucocytes, and by finding the appropriate bacterium.

Microscopical examination of brain and cord *post mortem* may sometimes be necessary. The differences between the changes found in anterior poliomyelitis and in lethargic encephalitis are demonstrated in the accompanying figures.

Differentiation from Epidemic or Lethargic Encephalitis.

Lethargic encephalitis affects apparently every age group and has occasionally to be differentiated

in children. Apart from the cerebro-spinal fluid findings this must be mainly on clinical grounds, but the experimental work with the virus of epidemic encephalitis has reached an interesting stage. It is not possible for me to give you a critical appreciation of the results of numerous workers in this field. Suffice it to say that though Muir, for example, held the view (1924) that the disease had been transmitted to monkeys and rabbits (by James McIntosh and others), yet we find that Simon Flexner who was responsible for so much of the experimental work with the virus of poliomyelitis, admitted (1923) his failure to transmit encephalitis after many attempts in monkeys and rabbits. He hints at a possible confusion by some workers with the virus of febrile herpes which is readily communicable and may cause one form of encephalitis in rabbits. He also points to the work of McCartney who found lesions resulting from a spontaneous encephalitis to be quite common in domestic rabbits. "It would, in my opinion," he writes, "be very unfortunate should we adopt a premature conviction regarding the ætiology of the disease."

Finally, we have had in Australia an epidemic with peculiar features investigated by Cleland and called by him "X" disease. It seems not improbable that this disease will recur and it may still be possible to elucidate its relation to the more common types of polio-encephalo-myelitis by a confirmatory investigation both histological and experimental and it is to be hoped that the Board of Health will make adequate provision for such an investigation.

THE EPIDEMIOLOGY AND ADMINISTRATIVE CONTROL OF ANTERIOR POLIOMYELITIS.[1]

By Robert Dick, M.B., Ch.M. (Sydney), D.P.H. (Cantab.),
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Poliomyelitis is a systemic infectious disease due to a specific filtrable virus and, so far as is known, man is the only carrier and distributor of the infection. Whilst the term infantile paralysis is more or less satisfactory for general descriptive purposes, it does not allow for the fact which is generally accepted, that many cases of poliomyelitis occur without the presence at any stage of the illness of any signs of paralysis. Moreover, the disease is not confined to infants.

Epidemiology.

History.

It would appear that the disease has affected human beings from very early times. The first mention of it in England was about the year 1784. During the early years of the nineteenth century cases of the disease were recorded in Great Britain and Europe and in 1840 Heine wrote a monograph on the paralyses met with in infants who had suffered from it. Later on in the century the disease appeared in more pronounced epidemic form, notably in Scandinavia, where a great deal of investigational work has been done on the subject. During the present century and particularly from about 1915 onwards there have been serious and widespread epidemics in various parts of the world. The United States of America may be cited as having been seriously affected.

As regards the history of the disease in Australia, Cleland and Ferguson contributed an article to the Australasian Medical Congress of 1914 from which it is learnt that the first recorded outbreak in Australia occurred in 1895, at Port Lincoln, South Australia, when there were fourteeen cases met with during March and April. None of these were fatal. In 1903 to 1904 during the summer months there was a rather extensive epidemic which spread through all the Australian States. Dr. R. B. Wade reported on thirty-four cases in the *Australasian Medical Gazette*, no fatalities occurred, and the late W. F. Litchfield reported on thirty-five cases. In 1908 there was an epidemic in Victoria involving one hundred and thirty-five patients with a fatality rate of 4.5%. In 1909 Dr. Macdonald Gill reported on twelve cases occurring in Sydney. In the same year there were a number of cases in the country districts of New South Wales. A series of twenty in the Tweed and Richmond River districts were reported upon by the late C. S. Willis.

Cases of the disease were recorded in the other States during 1909 and also in 1911 and 1912. In 1912 the disease was made a notifiable one in New

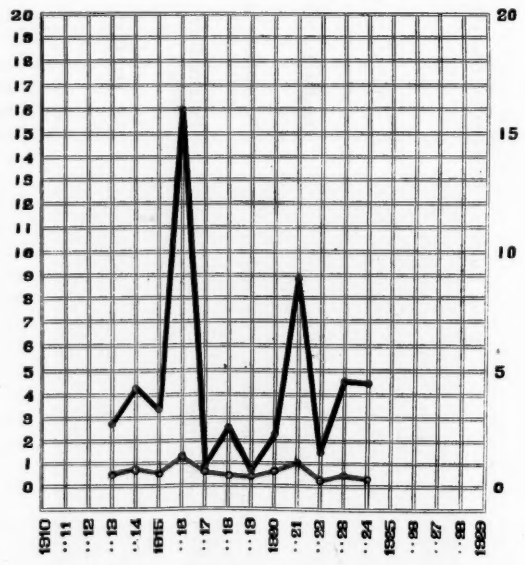

Figure I.
Showing Cases of Anterior Poliomyelitis Reported to Department of Public Health, New South Wales, from 1912. The heavy line represents the number of cases per 100,000 of mean population. The shaded line represents the number of deaths per 100,000 of mean population.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 7, 1925.

TABLE I.

COUNTRY	CENSUS POPULATION IN MILLIONS	CASES NOTIFIED 1919	1920	1921	1922	1923	1924	1925
ENGLAND & WALES	37¾	553	293	488	355	587	632	
U.S.A. 27 STATES	66¾	1790	2225	5880	2034	2912	2584	
U.S.A. PACIFIC STATES	5½	45	88	696	86	280	450	
NEW ZEALAND	1¼	11	76	267	98	16	58	970
AUSTRALIA	5½	135	71	236	116	136	229	

South Wales for the first time. The numbers of cases coming to the knowledge of the Health Department from that date down to the present time are shown in graph in Figure I.

Coming down to the present reports show that in those parts of the world where the disease has been usually much in evidence, its incidence is again on the increase, but its virulence is not so great as it was in 1911, 1912 and 1916.

Table I. shows the number of cases of poliomyelitis reported in England and Wales, the United States (twenty-seven States and the three States on the Pacific coast) and in Australia and New Zealand during the past six years, together with the available data for 1925.

Incubation Period.

The incubation period is not definitely settled; it is stated to vary from one to four days. In experiments on monkeys the incubation period has varied from three days to two weeks. The period may vary in different epidemics.

Age Incidence.

Although no age is immune, about 80% of all cases occur during childhood. Infants under one year contract the disease though not so often as slightly older children; breast feeding of infants has conferred no protection compared with bottle-feeding.

The great majority of cases occur in children under six years of age. It appears, however, that during some epidemics in which the majority of the susceptible population is attacked, the proportion of adults may be high. For example, in the epidemic on the Island of Nauru[1] with a population of two thousand five hundred there were seven hundred cases, the large majority of the patients being adults.

In the New Zealand epidemic of 1916, children under nine years of age formed the bulk of the patients. The heaviest incidence occurred in the age-group one to two years (8‰ children living at that age), followed by the group two to three years (6‰) and others in declining rates. In the present New Zealand outbreak children between two and three years appear to have been most frequently attacked. In the New South Wales epidemic of 1916, 29% of the cases occurred in children under one year and nearly 50% of the patients were children between one and four years. In the 1921 (New South Wales) epidemic only 7% of the cases occurred among children under one year, and over 57% among those between one and four years.

Fatality Rate.

The fatality rate varies in different epidemics from 5% to 15% or higher. In the New South Wales epidemic of 1916 it was 6.75% and in 1921 it was 11.9%. So far as has been ascertained the fatality rate of the recent New Zealand epidemic is about 13%.

Seasonal and Climatological Influences.

Warm weather, if not a predisposing cause of the disease, is its usual concomitant (see accompanying figures). Most of the epidemics recorded in the United States have occurred during the warm months. It should be noted, however, that outbreaks of epidemic proportions have occurred in cold countries like Norway and Sweden in the winter season.

The New Zealand epidemics of 1914 and 1916 both commenced about the beginning of autumn. The 1916 outbreak was more extensive and of greater severity than that of 1914, but the progress of each was almost identical. Commencing in the first week of February the epidemic reached its highest point at the end of March, thereafter falling with minor fluctuations to zero in the first week in June and after this date a very slight rise again occurred.

The New South Wales epidemic of 1916 ran a similar course except that it commenced earlier in December, 1915, reached its highest point in March and faded away by June. The 1921 epidemic had a sharper rise, reaching its maximum in February and declining earlier. This is shown in the accompanying graphs.

It is difficult to say precisely what are the special conditions met with in summer and autumn which favour the spread of the disease. The question of insect life which is usually in evidence during summer and autumn, will be referred to later.

It has been noted in certain countries that: (i.) High temperatures and a fairly low rainfall marked the epidemic period; (ii.) the fall in the severity of the epidemic synchronized very closely with the decline in the temperature; (iii.) the average temperatures of the period during which the epidemic lasted were higher, in some cases much higher, than the averages for the corresponding months of the preceding years. These conditions were noted in New Zealand during the 1916 epidemic.

Whatever other factors are necessary for the spread and propagation of the disease, a warm, dry season provides the conditions which are favourable to its establishment in epidemic form.

Sex.

It is usually stated that more males than females are affected. If the notification figures are taken as a guide, this is so, but the disparity is not very great and it is probably accounted for by the preponderance of males over females at all ages in the general population.

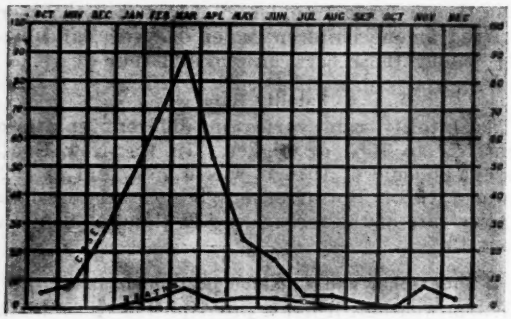

FIGURE II.

Showing Incidence of Infantile Paralysis in New South Wales Epidemic, 1915-1916. The cases numbered three hundred and eleven and the deaths twenty-one.

Infectivity.

The incidence of the disease does not as a rule bear any definite relation to the density of population amongst which it occurs. The better class districts usually suffer just as severely as the poorer quarters. It has been noted in various countries that epidemics are more severe in small villages and rural communities than in the larger centres. This has been remarked on in the present New Zealand outbreak.

In New South Wales, though the large centres of population have furnished the greatest number of cases, on analysis it is found that the less densely populated suburbs usually furnish a greater toll than do the more crowded centres of the city itself. It is unusual to have more than one case in a house, even though the fatality rate of the epidemic is a proportionately high one. Some writers aver that the disease has appeared successively for two or three years in the same house. Schools were early held responsible as potent factors in the dissemination of the disease. Generally speaking, when once a locality has suffered from the disease in epidemic form, few if any cases occur during the following year.

In this connexion Vaughan remarks on certain uniform characteristics of the disease noted in epidemics in various countries. First, that the disease instead of involving at one time a large and continuous area is dotted here and there, one, two or three cases in one centre and a similar occurrence happens some miles distant.

Taking into account only recognized cases it has been found in the majority of instances impossible to trace any connexion between the cases. Another characteristic is that after having visited one region, even in a scattered way, it apparently spares the same locality for a period of one to four years thereafter. One is practically forced to the conclusion that the number of persons susceptible to the disease in any community is small. But Vaughan points out that one should be cautious in accepting this statement. It may be that all the apparent non-susceptibles in a given community are really immune and owe this immunity to having had at some previous time an unrecognized attack of the disease. He remarks that "acute poliomyelitis is either a disease to which only a very small percentage of people are susceptible or it is a disease to which practically all are susceptible." In urban districts the disease is confined largely to children under five years. In rural epidemics it has happened that 50% of the cases were in adults. One explanation of this difference in the age incidence is that the higher age groups are not involved in epidemics, because they have had the disease and are immune. In Vaughan's opinion poliomyelitis is endemic the world over and shows itself in epidemic form only when by some accident enough of the population have escaped the disease in infancy to acquire it in later life. The behaviour of this disease as regards its heavier incidence amongst rural communities as compared with similar elements of the population living in cities, is somewhat akin to that of diphtheria, as has been recently pointed out by Zingher and Dudley in regard to the latter disease.

Reference may here be made to the results of Zingher in regard to the Schick test carried out by him in cases of poliomyelitis. He found that whereas amongst normal children aged one to four years, 32% of them gave a Schick reaction, nearly three times as many of the children who had suffered from poliomyelitis, gave a response to the Schick test. Zingher considers that a "susceptibility to one of the less contagious disease like poliomyelitis indicates that the child is more apt to be susceptible to other contagious and infectious diseases."

Mode of Spread.

The infecting agent or virus is found in the nervous system, in the secretions of nose and throat and possibly in the fæces. Discharges from the nose and throat have been used in experimental transmission of disease to monkeys and it is considered that this is the usual source of infection from one infected human being to another. Doubt

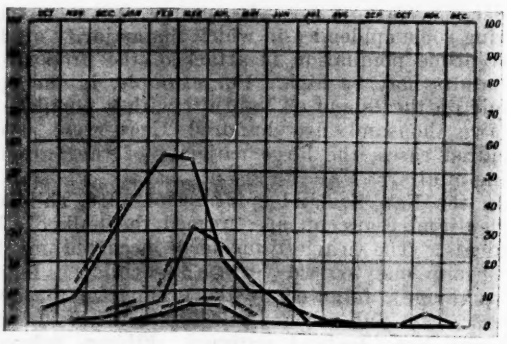

FIGURE III.

Showing Incidence of Infantile Paralysis in New South Wales Epidemic, 1915-1916, according to Districts. The upper line represents the Metropolitan District, the lowest line represents the Hunter River District and the middle line represents the remainder of the State.

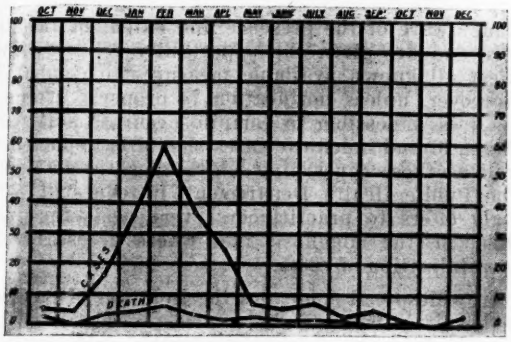

FIGURE IV.
Showing Incidence of Infantile Paralysis in New South Wales Epidemic, 1920-1921. The cases numbered one hundred and eighty-four and the deaths twenty-two.

has been expressed on this point by some writers in view of the fact that profuse secretions from the mucous membranes are not common in poliomyelitis. Vaughan inclines to the view that the virus is more frequently introduced through the digestive tract. The infection has not been found in any organs of the body apart from the nervous tissues except in rare cases. It has not been found in the blood of infected persons. Experimentally large quantities of blood from an infected monkey when injected into another healthy monkey have not produced an infection. This appears to put out of court the probability of any biting insect being the active agent in the spread of the disease. Although some experiments have proved that under certain circumstances the disease has been transferred from monkey to monkey by means of the stable fly bite, the conditions of the experiment were so artificial that any conclusion drawn from them must be accepted with a good deal of caution. However, Stomoxys does not as a rule bite human beings. If it be allowed that this fly does so occasionally, the amount of blood abstracted by it is comparatively small and it has been shown that even large quantities of blood produce no results. Robb who investigated this subject, states: "After an exhaustive inquiry the stable fly has now left the court without this stain upon its character."

The fact that epidemics occur in winter would also seem to exclude the probability of flies playing an important rôle in the transmission of the disease. There is no evidence to prove that bugs or lice are responsible for conveyance of the infection. Foodstuffs do not appear to act as the means of spread. There is no evidence that domestic animals play a part in transmitting the disease and though these animals are subject to various forms of disease displaying paralytic signs, it has not been shown that there is any relation between them and acute poliomyelitis.

It has been suggested by Brues that rats may perhaps act as a reservoir for the virus. If that were proved to be the case, the transmission of the disease by fleas or through contamination of foodstuffs would need to be considered.

As often happens where ignorance prevails almost every circumstance has been considered at various times to have some causal connexion or relationship with the disease; for example, during the New South Wales epidemic of 1916 numerous theories were put forward in the daily press as to the causation of the disease, according to one of which sea bathing was regarded as a likely cause.

Contacts.

It has been found in certain outbreaks that the incidence of the disease among contacts occurs in an irregular fashion; very close association, as for instance a child attacked by the disease occupying the same bed with other persons, often failed to transmit the disease to them. On the other hand instances have occurred where the disease has appeared to be highly communicable.

Abortive Forms and Carriers.

There is a possibility that persons suffering from abortive forms and carriers who are otherwise healthy, may harbour the virus and be able to transmit it to others.

The usual experience during epidemics is for the disease to manifest much diversity in the degree and extent of the paralysis. There may be extensive and permanent paralysis, paralysis which is

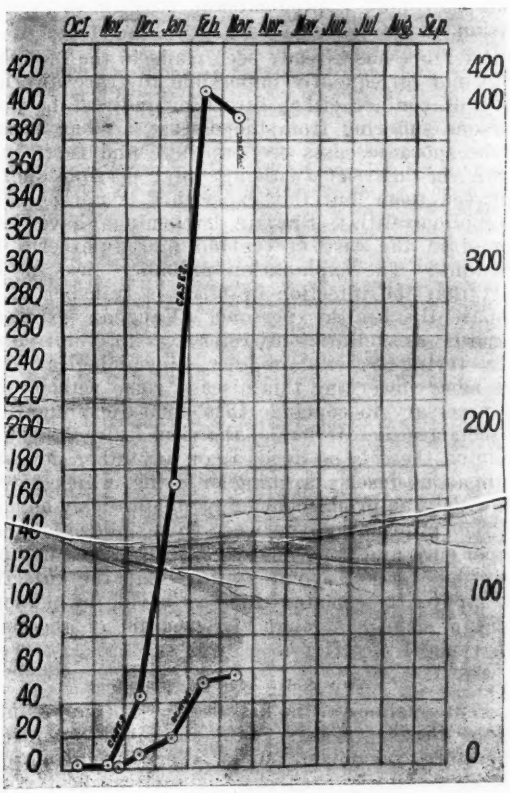

FIGURE V.
Showing Incidence of Infantile Paralysis in New Zealand Epidemic, 1924-25.

permanent but of slight extent or cases in which no signs of paralysis are present at all.

The diagnosis in the last mentioned variety may be questioned but there is a growing consensus of opinion that cases of the disease are often missed owing to the absence of the characteristic paralysis and that these constitute a far larger number in an epidemic than those with paralysis.

These mild and abortive cases probably do not figure in the statistics dealing with the disease. As a result of their experience in Sweden Kling and Wernstedt state that the virus carrier and the abortive cases in a family may be four or five times as numerous as those with typical symptoms.

It may be mentioned that monkeys inoculated with the virus of poliomyelitis occasionally pass through an indefinite illness without resultant paralysis, clinically similar to the abortive attacks observed in man.

Abortive cases in the Swedish epidemic of 1905 constituted 15% of the total according to Wickman. The mild and therefore probably missed cases are considered by Flexner to constitute the greater menace to public health.

So far as healthy carriers of the virus are concerned, Flexner states he is satisfied that they exist, but present knowledge does not allow us to say that they play a predominant part in the distribution of the disease.

Reference has already been made to the fact that it is not infrequently impossible to trace any history of contact either direct or indirect between persons suffering from the disease. What appear to be sporadic cases crop up here and there in a town or district without any possible nexus between them, but this experience is not peculiar to poliomyelitis. Similar happenings have been found in the case of typhoid and diphtheria for instance. The explanation probably lies in the fact that the infection or virus is transported by apparently healthy persons. Vaughan expresses himself as follows in reference to this point: "In trying to explain the epidemiological data we have concerning this disease, some persons find it necessary to conclude that domestic animals or insects are the carriers of the virus." In Vaughan's opinion there is no dog, cat or any other domestic animal, no insect, creeping or flying, which travels so widely, so promiscuously or ubiquitously as man.

The information derived from epidemiological experience and experimental research does not permit one to go beyond stating that the infection is apparently communicated as a rule by personal contact chiefly through the medium of the naso-pharyngeal secretions.

Administrative Control.

Acute poliomyelitis has been a notifiable infectious disease in this State since 1912 and it is therefore incumbent upon every medical practioner to notify each case as soon as he becomes aware of it.

According to the completeness with which notification is carried out, so are the health authorities made aware of the presence and extent of the disease and are given the opportunity to carry into effect all known available measures to combat it. Moreover, unless notification is properly attended to, it is impossible to compile accurate statistics. The mild and indefinite cases should be reported as well as those concerning which there is no doubt. The Public Health Department, if requested, willingly offers to practitioners every assistance possible in the diagnosis and where necessary the hospitalization of cases.

In widespread epidemics the isolation of patients in hospitals is no doubt a wise procedure, as the disease is more contagious during such periods. In the United States of America during the severe epidemic of 1916 strict quarantine was attempted, but the disease spread in spite of it. In more recent years less stringent measures have been put into operation.

In New South Wales the provisions of the *Health Acts* dealing with typhoid and similar notifiable diseases apply to acute poliomyelitis. Children of school age who have been attacked by the disease, are not allowed to return to school until a certificate is furnished by a medical practitioner that they are not liable to convey infection. School children who are contacts of a case of the disease, are excluded from school until twenty-one days after the paralysis first appeared in the patient.

It is recommended by some authorities that as a preventive the throats and nasal passages of contacts should be sprayed with a suitable mild disinfecting solution, such as potassium permanganate. All the patients' discharges, particularly those from nose and throat, should be carefully disinfected before being taken from the sick room.

Access of flies to the patient or his discharges should be prevented. Disinfection of utensils and objects soiled by the patient should be carried out in accordance with general principles and where the patient has been nursed at home there should be a terminal cleansing and disinfection of the house and its contents.

In virulent epidemics it may be necessary to close schools, cinemas and similar places where large numbers of children congregate in confined spaces.

There being no ready method available for the discovery of "carriers," it is impossible to exercise special control over them and as no effective method of immunization is yet available, the disease can only be dealt with on general principles. The injection of the serum of convalescent patients or of those who have recovered from a previous attack has been employed in the treatment of the disease and good results are reported to have followed its use in New Zealand.

This procedure would appear to offer some hope in combating the disease and by its early use the damaging effects of the virus upon the nerve centres may perhaps be obviated or minimized.

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Reports of Cases.

TWO CASES OF KATATONIC STUPOR.[1]

By E. J. T. THOMPSON, M.A. B.Sc., M.B., Ch.B. (Glasgow), *Resident Medical Officer, Hospital for Insane, Claremont, Western Australia.*

THE condition from which the two patients are suffering is that of *dementia præcox* katatonic stupor, showing the contrast with the condition of anergic stupor.

In the wards the patients are known as the twins, though they differ somewhat in age; Mrs. B. was forty-three years on admission three years ago and Mrs. L. twenty-eight two years ago. The usual age of onset is from twenty to thirty years. Though they differ so much in age, I do not think anyone would question that their conditions are similar.

Their histories are somewhat similar, the onset in each case having been sudden and having occurred during the puerperium. Both showed much depression with restlessness and refusal of food, but Mrs. B. is described as having had an attack of "incessant talking" about twelve years ago and as having attacked her mother with an axe. This I have no doubt was the real beginning of the disease and brings the time of onset to about the usual age. At the beginning of her present illness she expressed mixed delusions of grandeur and persecution, with auditory hallucinations. Mrs. L. was apparently mute from the beginning. Mrs. B. has a bad family history, her father having been "peculiar" before death at a fairly early age; her mother died a senile dement and her brothers and sisters ar described as highly strung and excitable. Mrs. L.'s heredity is unknown.

Both are good examples of negativism. They sit in the same position all day unless moved. The facial expression and whole attitude is fixed, the one usually with the expectation of heaven, the other with gloomier prospects. On admission both refused food; one had to be fed by tube for two to three months. Now they take food fairly well when it is placed in front of them, but neither would make any attempt to get food for themselves. They are dirty in their habits and saliva often flows from the mouth. Mrs. B. appparently is conscious of salivation as she is reported as having said: "I would be very much annoyed if any of my children did this, but I cannot help it."

Voluntary action is almost completely absent, but on rare occasions it manifests itself. Mrs. L. was once described as being violent and impulsive when she attacked a patient who was annoying her. Mrs. B. occasionally runs round the airing court in Marathon style and on one occasion when her arm was linked up with Mrs. L.'s, she dragged her round the court, Mrs. L. enjoying the joke and smiling most of the time.

[1] Read at the Annual Meeting of the Western Australian Branch of the British Medical Association on March 22, 1925.

Mutism is evident in both. Mrs. B. used to converse freely with her friends, but not with the Medical Officer; now she converses with no one. Mrs. L. I have heard speak on two occasions only, but in an unintelligible manner. She was reported a few nights ago as having said distinctly to a patient who was abusive towards a nurse: "If you can't speak to your equals, don't speak to your betters; you are no better than I am." (? inferiority complex.)

While it may seem trivial to note such small incidents, it is the only way in which we are able to find out whether perception is in abeyance or not. These patients will often cast a sly glance at the medical officer when talking to another patient and Mrs. L. on occasions laughs at some funny incidents in the wards if she thinks she is unobserved. After recovery or even during more lucid periods, a patient of this type can often tell what was said to her years before. The receptive centres are apparently not at fault, perception is acute and memory seems to be good. These are great differences from the anergic stupor.

It is difficult to say if mutism is present, whether or not the common sensations are disordered. Mrs. L. shows no reaction to temperature stimuli and pain. Mrs. B. reacts to pain at least, but their negativism is so complete that it forbids us to draw the conclusion that there is any disturbance of the sensory mechanism.

Their negativism is shown particularly when anything is being done for them. They resist forcibly any movements. While Mrs. L. shows merely passive resistance, Mrs. B. at times exhibits active negativism. If told to open her eyes widely, she will close them and *vice versa;* if told to put out her tongue, she will grip her lips tightly; if told to keep her hand by her side, she will raise it slowly above her head. Yet at times she shows automatic obedience. She has undressed herself when asked to by a nurse and has walked into her bath without resistance. Both are good examples of one of the most characteristic features of the condition—*flexibilitas cerea*—in which the limbs or body can be put in almost any position and will remain so for some considerable time. This is one of the main diagnostic features from the flaccid condition of anergic stupor.

This plastic condition in katatonia is interesting in view of the recent experiments of Royle and Hunter on plastic tonus. This condition seems to me to be the nearest approach we have in the human being to decerebrate rigidity. As Royle remarks: "The limbs of the decerebrate animal do not always remain extended, but may relax and take up a semi-flexed position. If the limb be now passively placed in extension, it will tend to remain in the extended position. These phenomena have been described by Sherrington as 'lengthening' and 'shortening' reactions." The general position of these patients is semi-flexion, but if the limb is passively placed in extension, the position is maintained for a considerable period and only gradually returns to semi-flexion.

Royle and Hunter have further demonstrated that the sympathetic plays an important part in maintaining plastic tone. In addition to the hypertonic condition of the muscles there is other evidence of sympatheticotonia in these patients, especially in Mrs. B.. The pulse is regular but rapid, the blood pressure is high (in Mrs. B. 170 millimetres systolic pressure, in Mrs. L. 155 millimetres systolic pressure), the pupils are slightly dilated and almost fixed. In Mrs. B. the palpebral fissures are widely open and there is the appearance of proptosis; the superficial and plantar reflexes are brisk and there is no clonus. In Mrs. B. the tendon reflexes are exaggerated and the knee jerks resemble the condition described by Royle in spastic conditions before the sympathetic is severed, that is the upward movement is brisk and the leg tends to remain in the extended position and relaxes more slowly. In Mrs. L. the tendon reflexes were exaggerated eight months ago and the skin was very dry and of the anserine or goose-skin type, but since then she has been on endocrine treatment reputed to be sympathetic depressant; the tendon reflexes are now dull, the skin about normal and her general condition has improved.

As in anergic stupor, *tache cérébrale* and dermographia are easily obtained.

From a psychological point of view the condition is regarded as an introversion. The patients at some time prior to the onset of illness have found themselves confronted with problems too big for their mental capacity. The conflict with the outside world thus set up they may be able to maintain as long as health aids them, but in periods of weakness as at child-birth or during the puerperium, they find themselves entirely unable to deal with it. Their only recourse is to shut themselves out from reality, barring the entrance of external stimuli to the level of consciousness and living in a dream world all their own.

The prognosis in these cases is more favourable than in any of the other forms of *dementia præcox*. Stoddart gives the figures: 60% end in extreme dementia, 27% in partial dementia, but sufficiently improved to be allowed to return home, 13% recover at least temporarily.

A CASE OF ANERGIC STUPOR.[1]

By J. Bentley, M.B., Ch.B. (Edinburgh),
Medical Superintendent, Hospital for the Insane, Claremont, Western Australia.

G.O., aged twenty-seven, a market gardener, was admitted to hospital on February 7, 1925.

Past History.

The patient was in the Italian Army during the war and was said to have been "gassed." Had acute rheumatism about two years ago and no other illnesses of any importance. No history of insanity or other nervous disease in the family was obtainable. The present attack began two weeks previous to admission, when he became very restless and troublesome and was taken to the Perth Hospital. During his stay in the Perth Hospital he was violent, restless, talkative, incoherent and noisy, refused food and had to be spoon fed. Dr. MacKenzie states that he was suffering from typical maniacal excitement.

Present Physical Condition.

The patient is poorly nourished. His pulse varies from sixty to seventy-eight and his temperature since admission has been subnormal. His systolic blood pressure is 90 millimetres of mercury. His extremities are cold, although the weather is hot. There is no œdema of the extremities. Patient is constipated and requires enemeta to produce evacuation of the bowels. Retention of urine was present shortly after admission and the average quantity drawn off by catheter daily was a little over twenty-two ounces. Examination of his chest reveals nothing abnormal, his knee jerks are absent and his plantar reflexes are not obtainable. His abdominal and cremasteric reflexes are active. There is a narrowing of the palpebral fissures, his pupils are of moderate size and are active to light, but his reaction to accommodation could not be tested. Pressure on the eyeballs produces a slowing of the pulse rate of eight per minute. It is difficult to say whether there is peripheral anæsthesia or not. The patient does not respond in any way to pin pricks. There is no muscular rigidity; the hands, feet and head if raised fall like a piece of stone when released. There is almost complete atonia of the voluntary muscles. On passing a catheter the urine only dribbled slowly and it was necessary to press on the abdomen to assist the atonic bladder. At the end of this pressure when the hand was removed, a hollow was seen to remain and it took several seconds for the abdomen to resume its normal contour.

Present Mental Condition.

On admission the patient was continually moaning. His attention could not be attracted and he did not reply to simple questions. At first he threw himself from one side of the bed to the other and would have fallen out if a sheet had not been placed across his chest to prevent him. Later he became perfectly still and apathetic. He lay in bed like a log and consciousness was at a very low ebb. He did not even respond to the passage of a catheter. Apparently he paid no attention to his surroundings. He showed no sign of recognition of his brother, nor did he respond in any way to remarks addressed to him in Italian. He resquires to be fed and will only take liquid nourishment. If solid food is placed in his mouth, he immediately spits it out.

Remarks.

This patient is suffering from typical anergic stupor. In this condition there is no ideation or perception and when the patients get well there is no memory of events occurring during their illness. These patients have to be spoon fed or sometimes tube fed, otherwise they would

Diagnosis.

The diagnosis has to be made from cerebral tumours, lethargic encephalitis, katatonic and melancholic stupor. The points to be considered include first the history. This patient had a sharp attack of acute mania and Stoddart states that anergic stupor may be primary or may frequently develope from melancholic or post-maniacal stupor. The absence of vomiting and of rise of temperature exclude tumour and *encephalitis lethargica*. The most difficult points in the diagnosis are to distinguish this condition from katatonic stupor. The history will help as katatonic stupor is preceded by a period of depression and the stupor may be followed by katatonic excitement, but excitement does not precede katatonic stupor. In katatonic stupor there is usually muscular rigidity, whereas this patient presents a condition of extreme flaccidity. In katatonic stupor the patient may be observed to take furtive glances, but in anergic stupor there is complete lack of all observeration.

Prognosis.

The prognosis in this case is good and recovery as a rule is complete (Stoddart). Patients recover in from three months to three years.

Treatment.

The chief aim in treatment should be feeding the patient and it is intended to try thyreoid extract in this condition.

It is interesting in view of the recent brilliant work of the late Professor Hunter to remark on the complete absence of plastic tone in this case and the condition of his eyes and the coldness of his limbs tend to show that this patient is in a vagotonic state.

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on March 22, 1925.

Reviews.

A HANDBOOK ON PSYCHIATRY.

W. S. Dawson's "Aids to Psychiatry" is a brilliant contribution to the students' aids series.[1] The author sets out "to present a concise description of the different forms of mental disorder" and succeeds admirably. The book is purely utilitarian in its object, "it emphasizes only the practical aspects of psychiatry." It is true that the author gives the teachings of the various schools of psychological medicine, but he does not seek to analyse these teachings, he does not criticize them. He has no new views of his own to publish. He has in short written for the medical student, for those who are learning to deal with cases of mental disorder and who are studying for a diploma in psychological medicine. The order of the book follows the well beaten path. The first chapter deals with normal psychology, the second with psycho-pathology and then the various forms of mental disorder are described on classical lines, their theories, pathology, clinical signs, diagnosis and treatment. Case taking and the legal aspect of insanity have not been forgotten. The book is thus a compendium of practically the whole of modern psychiatry.

[1] "Aids to Psychiatry," by W. S. Dawson, M.A., M.D. (Oxon.), M.R.C.P. (London), D.P.M.; 1924. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. viii. + 309. Price: 4s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 23, 1925.

The Macewen Memorial.

GREAT men are remembered more by the fruits of their work than by monuments erected by their contemporaries. Since, as Bacon has told us, knowledge is power, humanity is more indebted to those who contribute to the book of knowledge than to the most skilled technician whose work reaches but a segment of society. The true scientist lives a life of detachment of self; he neither seeks nor appreciates popularity and praise and is indifferent to the usual rewards that others claim for services rendered. When a great man passes hence, the few who have sat at his feet and listened to his teaching bewail their loss and mourn awhile; the larger world recognizing the value of his greatness creates a memorial to bear his name and to indicate to coming generations the honour in which he was held. Often the truth enunciated by him outlives its association with its discoverer and ultimately becomes so familiar to all students that the importance of its discovery is forgotten. For this reason the idea of the memorial is to be cherished.

Not long ago a great man made a pilgrimage to Australia and disseminated wisdom and encouragement among the members of the medical profession. Very shortly after his return to the old country Sir William Macewen journeyed to the Great Beyond. His work was known to all before he honoured the first session of the Australasian Medical Congress (British Medical Association) in Melbourne with his presence. Some had studied under him in Glasgow; the majority had read his numerous important contributions to knowledge; all had been taught his doctrines concerned with cerebral and spinal surgery, osteology, physiology and pathology. The courteous, stately gentleman whose keen intelligence and charming conversation arrested the attention of every member at that great gathering in Melbourne, has left a legacy to humanity that must be associated with his name as long as truth prevails. His work is finished; it is ours to use. Longfellow sang long ago:

> With thy rough ploughshare, Death, turn up the sod
> And spread the furrow for the seed we sow;
> This is the field and Acre of our God;
> This is the place where human harvests grow!

In Glasgow a committee has been formed for the purpose of creating a memorial to the late William Macewen, the father of cerebral surgery, the surgeon scientist. We have been asked by the honorary secretary of this committee, Doctor Archibald Young, to publish the details of the fund in Australia and to invite the members of the medical profession here to subscribe to it. William Macewen's last public service was his mission as ex-President of the British Medical Association to the congress of that association in Australia. The committee has resolved to ask for three thousand pounds to enable it to institute a threefold memorial. A bust of the great surgeon is to adorn the University of Glasgow and a replica is to be presented to Lady Macewen. A memorial lectureship is to be endowed and lastly a medal or prize in surgery is to be awarded each year. Medical practitioners in Australia will, we are convinced, be sufficiently large minded to associate themselves with this movement, notwithstanding the fact that the bust will be situated in Glasgow, the lectures will be delivered at the same place and the medal or prize will be awarded thirteen thousand miles away. All three monuments will remain within the great Empire and in these days of luxurious and rapid travel all will be accessible to Australian practitioners. The list of subscriptions just to hand announces the collection of £1,550. Sums ranging from one hundred guineas to ten shillings have been sent from far and wide. There is one subscription from New Zealand. In order to put Australia on the list, we have decided to collect subscriptions and forward them to Glasgow. Members who approve of the proposed memorial and wish to associate themselves with it, are invited to forward their contributions to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, "The Printing House," Seamer Street, Glebe, New South Wales. Exchange should be added to cheques when it is payable.

Acknowledgment of the receipt of contributions will be made in our columns. We who recognize the importance of William Macewen's contribution to medical knowledge, must give a tangible form to our tribute.

Current Comment.

BLOOD CHANGES IN CARCINOMA OF THE BONE MARROW.

THE blood picture commonly associated with malignant disease is as a rule frequently altered to a considerable extent when metastatic deposits involve the bone marrow. A disturbance of the reticulum takes place and marrow cells escape into the circulation. The blood picture may assume various forms. The simplest change is that which characterizes a severe secondary anæmia. The appearance, however, may be changed to such an extent that a differentiation from pernicious anæmia or leuchæmia becomes difficult. The erythrocytes may be reduced in number to as low a level as one and a half million and the colour index may be high. Polychromasia and basophilia are frequently seen, normoblasts may be present and sometimes megaloblasts. It has been pointed out that the degree of anæmia is not so severe as in pernicious anæmia, but that the qualitative changes are more prominent in the early stages than in cases of pernicious anæmia with similar cell counts. In the majority of cases of this nature a relative increase of polymorpho-nuclear neutrophile leucocytes is seen together with some myelocytes. When the picture resembles leuchæmia, great variation is seen. The leucocytes may reach a total of anything up to one hundred thousand per cubic millimetre, while myeloblasts, myelocytes and an excess of eosinophile cells have been found.

In the course of his work on carcinoma of the bone marrow in 1922, to which reference was made in this journal in February, 1923, Piney made some important observations on the blood changes associated with this condition. He pointed out in detail the differences between the blood pictures of pernicious anæmia and the pseudo-pernicious anæmia associated with carcinoma of the marrow. He held that in the latter condition there was evidence of a grave disturbance of the erythropoietic organs and that in addition there appeared to be some interference of a stimulating nature acting on the leucopoietic mechanism. He assumed that the interference with the erythropoietic system was of the nature of trauma from the growth of deposits. By analogy with the changes produced in the condition of the blood by an extravascular lesion in Hodgkin's disease, he concluded that the changes occurring in the blood in malignant disease pointed to an intravascular lesion. The progressive anæmia of malignant disease in his opinion leads to an increase in the amount of red marrow present in the bones and this results in further extension of deposits. He admitted that cases of carcinosis of the marrow occur in which the pseudo-pernicious blood picture is absent and claimed that if the existence of idiopathic changes of marrow aplasia in these malignant conditions could not be admitted, it was possible to conjecture as to the cause of the absence of the peculiar blood picture. In gelatinous degeneration of the marrow it would be surprising if much evidence of reaction to any form of lesion were present. If metastases of cancer settled in the bone marrow at an early stage of the life history of the primary growth, it would not be expected that blood changes would be very extensive owing to the absence of hyperplasia due to lack of previous anæmia. Piney held that a third cause of the absence of pseudo-pernicious changes would be the great extension of the cancerous process and extreme destruction of the marrow tissue. He concluded that the effect of deposits of cancer in the bone marrow is specific and that there are apparently insuperable difficulties about any explanation of the blood changes if the conception of an intravascular trauma be not accepted. The pseudo-pernicious blood changes occur in those places in which the disease has been present for a sufficiently long time to permit hyperplasia of the marrow to take place.

An interesting report has recently been made by Sir George Thomas Beatson on a case of osseous metastasis from primary carcinoma of the right breast.[1] The blood picture is of sufficient interest for reproduction here in full. The erythrocytes numbered 3,500,000 per cubic millimetre, the hæmoglobin value was 70% and the colour index was one. The white cells numbered 70,000 per cubic millimetre and of these 77% were neutrophile cells, 1% eosinophile cells, 3% transitional cells and 4% myelocytes. Small mononuclear cells contributed 11% and large mononuclears 4% of the leucocytes. This picture is one of secondary anæmia and leucocytosis. The patient was fifty-five years of age and the condition was one of long standing, six years prior to admission to hospital. At *post mortem* examination almost every bone in the body with the exception of the sternum was found to be extensively involved with carcinomatous deposit. Sir George Beatson asks what the blood picture signified, whether it reflected the condition of the bones and, if so, what variations did the formed elements undergo. In reply he states that distinct anatomical changes were present in the blood, but that they could not be said to coincide with the osseous lesions. He points out that on examination of the blood no nucleated red corpuscles and no megalocytes were found and that in no way was the condition in keeping with Piney's "metastatic anæmia of osseous carcinosis." He refers to the views of Harrington and Peacher that the recorded cases of osseous carcinosis fall into two groups, one characterized by no special changes in the blood

[1] *The British Journal of Surgery*, January, 1925.

and the other by variations suggestive of pernicious anæmia. He assigns the condition found in his patient to the first group. In the absence of ulceration of the primary growth, sepsis and hæmorrhage no erythroblastic reaction had occurred. Red marrow was absent from the epiphyseal ends of the humerus and femur and the small amount of red marrow present was seen in the shaft of the bone.

It is interesting to consider these observations in conjunction with Piney's teaching. Piney lays great stress on what he calls an intravascular trauma in the production of blood changes in metastatic carcinosis of bone. He uses this view in justification of his thesis on the vascular rather than the lymphatic spread of bone metastases. From the *post mortem* findings of Sir George Beatson's case the majority of observers will agree with him that the secondary deposits were blood borne. The history of the patient's illness was a long one and ample time would be allowed for the production of an erythropoietic reaction. It would be expected, if Piney's views are accepted, that the patient's blood would have manifested the picture described as that of pseudo-pernicious anæmia. This, however, was not the case. Piney gives as one reason for the absence of pseudo-pernicious changes the great extension of the cancerous process and extreme destruction of marrow tissue. Piney did not state whether he would expect in such a case that the blood would pass through a pseudo-pernicious phase. Presumably he would. Examination of the blood of Sir George Beatson's patient was apparently not undertaken until the disease was well advanced. Further researches will possibly reveal the nature of the condition necessary for the production of the pseudo-pernicious blood changes. Research of this nature will be of interest also in connexion with the pseudo-leuchæmic changes associated with bone cancer in view of the fact that many observers regard the various forms of leuchæmia as being neoplastic in nature.

ABERRANT PANCREATIC TISSUE.

THE pancreas appears during the fourth week of embryonic life in the form of two or maybe three processes of hypoblastic tissue from that part of the embryonic gut which subsequently forms the second part of the duodenum. Of the two buds the smaller arises from the ventral aspect of the duodenum in conjunction with the hepatic diverticulum and forms the lower part of the head of the pancreas. The greater part of this organ is formed from a process which arises from the dorsal aspect of the duodenum nearer the stomach than the ventral process. The larger portion grows into the dorsal mesogastrium towards the spleen and unites in the mesogastrium with the ventral bud. The ducts of both processes may persist. A third pancreatic bud has been observed in the human embryo. It arises from the ventral aspect of the gut and corresponds to a third bud found in the developing pancreas of lower vertebrates. It will be seen that in this developing process small fragments may easily become separated from the hollow cylindrical processes as they divide and redivide and become implanted in a neighbouring organ where their development may proceed in an apparently normal manner. Similar aberrant forms are seen in other parts of the body. Thus occasionally islands of thyreoid tissue may be found in the vicinity of the thyreoid gland.

Lieutenant R. M. Choisser has recently reported an instance of aberrant pancreas which is not without surgical interest.[1] The patient was an ex-service man, aged fifty-seven years. He complained for about eighteen months of continuous pain in the upper part of the abdomen and of loss of strength. He was much emaciated. After X-ray examination a diagnosis of malignant disease of the pylorus was made. Gastro-enterostomy was performed and the patient subsequently died. At *post mortem* examination a tumour mass measuring one and a half by two centimetres was found projecting into the pylorus from beneath the mucosa. The mass was of firm consistency, whitish in appearance and surrounded by a fibrous capsule. Two small, bile stained ducts were found coming from the base of the mass. These anastomosed and formed one duct which emptied into one of the hepatic ducts. On microscopical examination the tumour was found to consist of pancreatic acini, fibrous tissue and ducts of various sizes. In one area a small mass of islet cells was found. No sign of malignant disease was discovered. Mr. Choisser lays stress on the clinical significance of this condition and discusses the hypotheses concerning the ætiology of these aberrant forms.

In the first place he refers to the development of the pancreas and to the view already expressed of the separation of fragments from the growing processes. He states that this is not generally accepted, for if it were true, the masses would be large and would always be found in the gastro-intestinal tract. He has collected eighty-one cases from the literature and finds that this is not so. The Cohnheim theory of embryonic rests does not apply, as sections of the masses reveal in the majority of cases mature differentiated acinic cells and well developed independent ducts. Zenker believes that these structures originate in the embryo by the formation of additional duodenal diverticula which continue to develope singly and are carried along the gastro-intestinal tract as it increases in length. Mr. Choisser states that this theory may at first seem reasonable and that it accounts for aberrant masses in the stomach and intestine, but that it cannot explain their existence in such places as the spleen, mesentery and omentum. It is obvious that any hypothesis must be entirely speculative and Mr. Choisser is in accord with that propounded by Horgan. Horgan believes that the dorsal and ventral *Anlagen* of the pancreas are out of proportion to the size of the original gut and that during development they come into contact with some developing viscus and are grafted thereon.

[1] *United States Naval Medical Bulletin*, March, 1925.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Cystic Malformation of the Lung.

E. CAUTLEY (*British Journal of Children's Diseases*, April-June, 1924) reports the case of a cystic malformation of the lung in a male infant who died at the age of nineteen weeks. He was admitted to hospital with a history that the breathing became distressed and the chest more prominent one month previously. A week before admission he became worse, having attacks of cyanosis. The breathing was rapid, the chest prominent and there was absence of air entry over the right lung. He died ten days after admission. On opening the chest at necropsy a large, red cyst was found filling the right side of the thorax and extending to the left side of the sternum. It collapsed on puncture and only contained air. Its posterior wall was formed by reticulated lung tissue which was airless. The right lower lobe was collapsed. The upper lobe was represented by a small tongue-like process containing air. The flattened reticulated basis of the tumour was lung tissue, probably representative of portions of the upper middle lobes. The bronchi were patent, but no communication with the air-cyst was found. The author thinks it probable that originally there was a communication between the cyst and the bronchus and that the onset of symptoms six weeks before death was due to the closure of this orifice.

Chorea.

C. J. MACALISTER (*Proceedings of the Royal Society of Medicine*, November, 1924) discusses the results of an investigation with reference to chorea. He classifies chorea into the following types: (i.) The ordinary rheumatic or toxic types, (ii.) stock-brained cases related to an inherited tendency (iii.) a climacteric type. He first distinguishes chorea which is rheumatic in origin or in which endo- or pericarditis occurs from that in which such is not the case. The latter type is often ascribed to fright or mental upset or to some peripheral irritation. Inquiry into the histories of patients suffering from both types revealed the fact that in chorea caused by fright *et cetera* the children were sometimes left-handed themselves or if right-handed, came of a left-handed stock. In the history of patients with the rheumatic form fright was only occasionally recorded and probably represented rheumatism in children with the inherited tendency. Stammering occasionally occurred in the families of patients suffering from the stock-brained type. The writer regards the choreic movements as representing a gross motor ataxia analogous to vocal stammering. This chorea probably did not occur in children of a purely right-handed strain, nor would it occur in those of a purely left-handed strain, but left-handedness uninfluenced by a right-handed inheritance must be extremely rare. The writer finds the explanation of these cases in a partial or repressed transposition of cerebral function. As a consequence of fright or emotion or peripheral disturbances an unbalancing of coordination was brought about by some unusual relation of function on the opposite sides of the brain. In referring to non-rheumatic chorea occurring at or about the time of puberty, the writer states that these conditions are due to a chemical or metabolic disturbance as distinct from the anatomical incoordination already mentioned.

Xanthoma Tuberosum.

G. A. HARRISON (*Proceedings of the Royal Society of Medicine*, November, 1924) reports a case of *xanthoma tuberosum* occurring in a girl aged eight years and ten months. Xanthoma nodules were present on the right elbow and right knee. A larger nodule on the left elbow had been excised for examination. This nodule was the first to appear, three years previously. Hypercholesterolæmia and an excess of cholesterol in the deposit in the skin were present. The writer has the patient under investigation, with the object of determining whether the diabetic and non-diabetic varieties of xanthoma are different grades of the same condition or are separate diseases and whether disorder of the liver or pancreas or both or of some other organ or organs is responsible for xanthoma. This patient has a normal blood sugar curve after fifty grammes of dextrose. The urine is normal and no response is obtained to the Van den Bergh test. There is no naked eye lipæmia and the fæces are normal both macroscopically and microscopically. The condition, therefore, belongs to the non-diabetic group. With regard to treatment the writer proposes to try the effect of "Insulin" when the patient is in hospital, where an ample carbo-hydrate intake can be assured and the necessary determination of blood sugar and blood cholesterol can be made.

Fat in the Diet of Infants.

J. P. CROZER GRIFFITH (*Atlantic Medical Journal*, January, 1925) discusses the need for fat in the infant's diet and refers also to the disadvantages of an excess of this constituent in the food. Even in the normal infant fat is liable to cause trouble, unless its administration is carefully managed and in babies already ill with gastro-intestinal disturbances it often makes the condition worse. While in theory the infant should be able to take 4% of fat, as found in human milk, in practice this is not the case. Probably one reason is the high percentage of volatile fatty acids present in cow's milk. Vomiting and disturbance of the stools occur from fat indigestion. The exudative diathesis is particularly liable to occur in children who have been overfed, especially with butter fat. In the chronic intestinal indigestion of infants and of older children fat is one of the most difficult elements to digest. The influence of fat unguarded by the proper amount of carbo-hydrate in producing acidosis in older subjects is well recognized and this is true of infants also. The need for an adequate amount of fat is becoming more apparent with the advance of knowledge. It is not a matter of indifference whether the calories come from fat or from carbo-hydrate. The substitution of fat to a certain extent by protein is a wasteful physiological procedure. Protein should be used to build tissue, not to produce calories. Even 3% of bovine fat is difficult for many healthy infants to take without digestive disturbance. The writer refers to two formulæ which seem to him to fulfil requirements. The first is the lactic acid milk recommended by Marriott. In the preparation of this formula lactic acid (U.S.P.) and corn syrup are employed; the resulting food contains approximately 4% fat, 10% carbo-hydrate and 4% protein. The second formula is the butter flour mixture of Czerny and Kleidschmidt. By a process in which the volatile fatty acids are driven off by heat, and a browning of the butter and added flour, a mixture is obtained which contains about 4.6% fat, 8% carbo-hydrate and about 1.5% protein.

Cœliac Disease.

H. MAUTNER (*Klinische Wochenschrift*, January 22, 1925) describes a case of cœliac disease. The main symptoms were lack of development and abnormal whitish bulky stools. Definite anæmia was noted and great increase in appetite and thirst were present. Œdema was present together with abdominal enlargement, with meteorism and increased size of both liver and spleen. Cases occur mainly in nervous children of the better classes. In his patient no treatment was successful until he injected horse serum. In other instances he had given pituitary extract with very good results at first, though relapse occurred within four months. *Post mortem* examination reveals no primary infection of the pancreas, though there are possibly secondary changes due to deficient food supplies analogous to the œdema noted during the war.

ORTHOPÆDIC SURGERY.

Autogenous Bone Grafts for Non-Union in Long Bones.

N. T. KIRK (*Journal of Bone and Joint Surgery*, October, 1924) reports the results of one hundred and fifty-eight autogenous bone grafts. The grafts were used in (i.) twenty-nine ununited simple fractures without infection, (ii.) one hundred and twenty-nine fractures following war wounds

or compound fractures followed by infection of bone and soft parts. In the first group twenty-seven grafts were successful. One failure was due to the graft being too small to withstand the muscle pull after correction of an old deformity. The second failure was in the femur which refractured after the seventh month. Results in the second group were quite different. Fifty-three out of one hundred and twenty-nine grafts were unsuccessful. The changes in the bones and other tissues as a result of prolonged sepsis or treatment was a cause of failure. Loss of substance was the rule in this group, the amount of bone missing varying up to twelve and a half centimetres. Scar incision was practised before bone grafting was attempted. The grafts used were at least three times the length of loss of substance or poor bone with which it was in contact. The use of sutures to hold the grafts was abandoned in favour of self-retaining grafts. Plaster of Paris was used as a splinting agent and was worn for months in most instances. Infection was the greatest cause of failure. Two sets of instruments were used, so that the healthy tibia might not be infected from the area to be grafted. Atrophy was apparently the cause of fifteen failures. Fracture of the graft occurred in a number of cases, some as late as the eighth month. All were due to trauma. During grafting operation the blood pressure is taken every ten to twenty minutes owing to the fact that shock is more easily produced than in an ordinary operation. The author found that the inner surface of the tibia from which the grafts were taken, was rapidly restored. In one instance he removed three grafts from the same place.

An Abduction Shoe-Wedge.

JOHN JOSEPH NUTT (*Journal of Bone and Joint Surgery*, October, 1924) draws attention to a method of wedging the soles of shoes in the treatment of an abducted foot. The wedge does not extend all along the inner side of the heel, but is placed at the front only. It tilts the *os calcis* upwards and backwards as well as outwards. Another wedge reaches from the supporting surface of the forepart of the shoe to a position under the first metatarsal. These wedges should not be used when dorsiflexion is limited to ninety degrees, nor without instructions in correct walking.

An Anatomical Variation of the Lumbo-Sacral Joint.

ARMITAGE WHITMAN (*Journal of Bone and Joint Surgery*, October, 1924) deals with observations upon the anatomical variation of the lumbo-sacral joint. The condition he describes, is one that has not been previously distinguished under those grouped as "low back pain" or chronic backache. The pain in these patients was fairly well localized in the region of the lumbo-sacral joint and often radiated down the front or back of the thighs. It is induced either by indirect violence such as a fall on the buttocks or the feet or by muscular effort such as an attempt to lift a heavy weight. Two patients stated that they were actually paralysed in the lower extremities. Pain may be lessened by some form of support. From the first appearance of the symptoms the patient recognizes that he is never completely well and he is always conscious that the back is weak. The patients described are of no definite physical type, but there was one characteristic that was prominent. Instead of an exaggerated curve of general lordosis, there is a sharp increase in the angle between the sacral and lumbar portions of the spine. The plane of the long axis of the sacrum may be almost horizontal. On running the fingers down the spinous process the hollow between the muscles in lumbar region is found to be much deeper and on passing over the tip of the fifth lumbar vertebra a sharp obstruction will be felt. This is the spinous process of the first sacral vertebra. Tenderness on pressure is usually present at the lumbo-sacral junction over the middle line. All movements may be restricted by pain and spasm. X-ray examination reveals a definite abnormal condition. The long axis of the sacrum lies almost in the horizontal plane so that the first sacral vertebra affords little or no support to the fifth lumbar vertebra. These characteristics really represent a mild degree of spondylolisthesis. The author reports five cases, in one of which a bone graft was used to cause a union between the last lumbar vertebra and the sacrum. This was successful. In other cases the spinal brace was employed.

The Treatment of Scoliosis.

R. W. LOVETT AND A. H. BREWSTER (*Journal of Bone and Joint Surgery*, October, 1924) present a report of progress in the method of treating scoliosis which employs a different application of force from that usually used. The method has proved more effective in securing rapid correction than any other form of treatment they have tried. They use the apex of the convexity of the curve as a fixed point and bend the spine above and below it on that as a fixed point. A hinged plaster of Paris jacket is employed and the hinge is placed opposite the apex of the curve. For the manufacture of the jacket the patient is suspended by a head sling and a close fitting plaster is applied from the axilla to below the greater trochanter. The jacket is immediately cut off by a vertical incision in front. From this a plaster of Paris model of the patient is obtained and on this model a plaster of Paris jacket is made. The jacket is divided transversely at the apex of the lateral curve and fastened together by "garden gate" hinges. On the other side a turnbuckle connects the two segments and is so arranged that it can be removed. The turnbuckle is used for stretching the patient once or twice a day and between the stretching periods the jacket is held in position by two locks. Gymnastics and manipulations of a heavy kind are regarded as a desirable part of this treatment.

Blood Viscosity in Trophic Disturbances of Circulation.

WALTER G. STERN (*Journal of Bone and Joint Surgery*, October, 1924) issues a preliminary report on the blood viscosity in trophic disturbances of the circulation. Under this title there exists a rather vague group of circulatory disturbances of unknown origin which are of interest to the orthopædic surgeon. Chief symptoms associated with these disturbances are pain and disability in the extremities together with coldness, numbness, cyanosis, loss of pulse, intermittent limping, painful papules and skin spots, ulcerations and gangrene. The author suggests the term angiotrophosis as a generic term to be applied to the entire group of symptoms. It is the belief of the author that these various symptom complexes are closely related to one another and that they only differ fundamentally in the severity of the toxic agent causing the disease and the susceptibility of the individual. It is beyond question that the Polish and Galician Jews, the Swedes in North America and the Japanese are more susceptible to this trouble and especially to the type called Buerger's disease, thrombo-angiitis or spontaneous gangrene. Three operations have been offered for the alleviation of symptoms of this condition, but have failed to gain universal recognition and have been discredited. The operation for the reversal of the circulation has been advised by Wieting and ligation of the femoral vein by Lilienthal, but neither procedure has commended itself to medical practitioners and both have been abandoned. Another operative cure has been advanced by Leriche who advocates periarterial sympathectomy. The most recent observation in these conditions is that of Mayesima. It is that in the majority there is a permanently high blood viscosity. Following upon this, Kogo and McArthur endeavoured to reduce the viscosity of the blood by giving large quantities of fluid. The author has directed his attention to measuring the viscosity of the blood in various conditions. He has made over four hundred and fifty observations of blood viscosity in healthy and various pathological conditions, but the only group with persistently high blood viscosity is the angiotrophotic group. Chilblains are not associated with a viscosity above the normal limits. A few patients with claw foot gave sufficient evidence of circulatory disturbances of the limb to lead him to estimate the blood viscosity. Of four patients examined, two had viscosity above the normal.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in conjunction with the Section of Hygiene and Preventive Medicine at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on April 7, 1925, DR. R. B. WADE, the President, in the chair.

Infantile Paralysis.

DR. C. P. B. CLUBBE read a paper entitled: "The Diagnosis and Clinical Features of Infantile Paralysis" (see page 527).

DR. A. H. TEBBUTT, D.S.O., read a paper entitled: "The Pathological Aspect of Infantile Paralysis" (see page 533).

DR. ROBERT DICK read a paper entitled: "The Epidemiologoy and Administrative Control of Infantile Paralysis" (see page 536).

DR. J. S. PURDY, D.S.O., said that unfortunately he had not been able to hear all the papers read that evening as he had to attend another function. During the previous months he had had the opportunity of following the epidemic in New Zealand. He had attended on February 3 a conference of one hundred doctors in Auckland, called to discuss the outbreak of infantile paralysis in the Dominion, as well as having had the opportunity of attending the New Zealand Medical Congress.

At Auckland Dr. Gilmore had given a *résumé* of the work of Flexner, Rosenau and others. The clinical features had been described by Dr. Williams, whilst the epidemiology had been dealt with by Dr. Hughes, the District Health Officer. Contributors to the discussion had included Sir Maui Pomare, a former colleague, now Minister of Health, Sir Frederick Truby King, Dr. P. H. Buck, the Native Health Officer, and others.

Dr. Purdy had pointed out the similarity of the graph showing the incidence in Auckland to the graph which he had published in the 1908 New Zealand health report of the 1908 outbreak of typhoid and made special reference to the fact that the records of the Auckland Hospital for the twenty years previous thereto had shown that typhoid in Auckland as to its highest incidence synchronized with the prevalence of flies, which were most numerous whenever the temperature was over 65° Fahrenheit.

He had suggested that the Government might put aside at least £3,000 to initiate a thorough medical research investigation into this and other diseases and that a prize of one hundred and fifty guineas should be offered for the essay with the most original contribution to the solution of the problem.

Dr. Purdy quoted from the summary of the report by Dr. Sydney Smith on the New Zealand epidemic in 1916 to the effect that infantile paralysis was a disease of which the habitat as far as was known, was man and that it was spread from actual patients and by carriers. He referred to the number of abortive cases. Dr. Purdy also made reference to the experimental work which had been carried out by Rosenau and by Wernstedt and Petterson, of the Medical Institute of Sweden. The general opinion was that the disease could be spread by direct contact from the sick to the well by naso-pharyngeal and buccal discharges or by discharge from the intestines or possibly according to some by an intermediary such as an insect.

Dr. Purdy related the incidence associated with the disease in Auckland in 1908 and in Tasmania in 1912, where there had been twelve cases in the upper Huon district. At that time having read the report of the International Dermographical Congress held at Washington in 1911, he had been impressed with the possibility of the disease being carried by the common stable biting fly (*Stomoxys calcitrans*). Richardson had published a report of the Board of Health, Massachusetts, of an investigation and had claimed the discovery of the so-called globoid bodies in the secretion obtained from patients. The same bodies had been found by Dr. Gilmore in Auckland and by Dr. Lynch in Wellington.

Wernstedt and Petterson, of Sweden, had demonstrated the transmission of the virus both from patients who had died of infantile paralysis and from the living, by secretions from the naso-pharynx and from the intestines, whilst the disease had been transmitted from an emulsion of the spinal cord. Rosenau claimed to have transmitted the disease from sick to well monkeys by the bite of the Stomoxys in six out of ten instances. Other observers, however, maintained that the disease as seen in monkeys was neither of the same type clinically nor pathologically as that seen in human beings.

Before leaving for New Zealand Mr. Clunies Ross, of the Veterinary Department, Sydney University, had written to Dr. Purdy and seen him in regard to the statement that the disease was transmitted by a tick. Mr. Ross had repudiated the allegations with regard to tick paralysis in dogs being similar to infantile paralysis in human beings. As a matter of fact tick paralysis in dogs was limited in Australia to the coastal belt, whereas infantile paralysis was more generally distributed, although not of course found in the tropics and tick paralysis in dogs was neither clinically nor pathologically akin to infantile paralysis.

On visiting the special ward of the Auckland Hospital where forty patients were under treatment, he had like others, been struck first of all by the fact that the children were of a well-nourished, healthy type and were generally fair and high coloured. As in the previous outbreaks, it was rare to have more than one case from a household, although there were instances of two in a household and one doctor claimed to have seen three patients who came from the same family.

Up to the time of Dr. Purdy's return from New Zealand there had only been one case among the 55,000 Maoris of the Dominion. Whilst some ascribed the immunity of the Maori possibly to pigment, Dr. Buck suggested that possibly it was due to the Maori not being so highly civilized as the *pakeha* (the white man).

It was remarkable that in the more congested and poorer part of Auckland, the Freeman's Bay area (corresponding to the Surry Hills area in Sydney), no case of the disease had been found, whereas in the outlying and more wealthy suburbs, such as Remuera, Mount Eden and Epsom, the disease was widely distributed. Unlike the New Zealand outbreak in 1916, the incidence had not followed the main lines of communication. At Whangarei Hospital he had seen three children who as pointed out by the medical superintendent, had come from three different points of the compass fully forty miles apart. In these cases they had to strain the carrier theory very considerably to explain the occurrence.

As a matter of fact he had heard a conversation on the telephone in which a doctor had suggested carriers and contacts and the reply to this had been "bunkum." Although Dr. Purdy did not think the carrier theory or contacts could be described as "bunkum" and that probably the main means of transmission was by human beings, yet he could explain certain phenomena only by transmission of the disease by some other intermediary.

At the special meeting of the New Zealand Branch of the British Medical Association at Auckland and again at the Congress at Mount Cook, Dr. Purdy had drawn attention to the seasonal incidence of the disease in New Zealand, in the upper Huon, Tasmania, in 1912 and in Sydney in 1916. In the lastnamed outbreak one half of the one hundred and eighty-six cases in the metropolitan area in that year had occurred in March and April. This period synchronized with the prevalence of the biting stable fly (*Stomoxys calcitrans*). He was surprised at the suggestion that this fly did not bite humans to any extent, as his experience as a surfer at Bondi beach was certainly to the contrary. Infection by this fly would explain the occurrence of the disease in the outlying suburbs of Auckland. In these localities stables and cow sheds were found and the practice of using horse manure for mulching gardens, especially for growing roses, made the presence of these flies more likely.

The chance infection suggested a comparatively non-ubiquitous insect carrier such as the stable fly. Trans-

mission by biting stable flies would also explain sporadic occurrence of the disease at considerable distances where no human contact could be traced or in regard to which the human carrier theory had to be stretched to a considerable extent.

Dr. Purdy passed around a letter from Dr. Tillyard, Entomologist, Cawthron Institute, Nelson, New Zealand, concurring with his suggestion that the *Stomoxys cal. citrans* was strongly incriminated as a carrier of this disease.

Dr. Purdy described the restrictions existing in New Zealand. The schools were closed. No children under sixteen years of age were permitted to travel on trains, trams, buses or ferries or to attend theatres, cinemas, picnics or any public gathering, even church services.

Epidemic disease, he remarked, however, was seldom checked by quarantine which was fast being reduced in older countries to mere isolation of affected individuals and actual contacts.

He would not presume to enter into the question of diagnosis, as the only occasion on which he had done so was in 1908. Whilst taking his youngster from his house in Princess Street through Central Park, Auckland, to the Health Office one Saturday afternoon, he had noticed the boy stumbling and on his return said to his mother: "I am afraid Cecil is developing anterior poliomyelitis." The mother being alarmed at so formidable a name, rushed to undress the boy only to find that the stumbling had been due to his two legs having been put through one trouser leg.

Dr. Harvey Sutton, O.B.E., said that infantile paralysis was a very old disease. Osler considered that Mephibosheth had been the subject of infantile paralysis and Sir Walter Scott had undoubtedly been affected by it. Insect life with its associations was one of the features of modern public health. He was, however, a little sceptic in regard to Stomoxys and its relationship to infantile paralysis. Dr. Sutton knew from personal experience at Rose Bay that it could bite man and bite him most effectively. He pointed out that a definite rise occurred during November and December in the incidence of enteric fever which was a fly-borne disease. In connexion with another fly-borne disease, epidemic ophthalmia, due to the Koch-Weeks bacillus, two rises occurred, one in October and November and the other at about April. Why was the first peak absent in the graph of the incidence of infantile paralysis? Warm climate appeared to predispose to epidemics. Scotland was affected less than England and Wales by the disease; its climate was colder. In New Zealand the infection appeared to spread from north to south. If curves of the March-April incidence were to be trusted at all, Australia was not going to suffer from an epidemic of infantile paralysis during 1925. In 1926, however, the periodical rise of 1916 and 1921 was due to recur and an epidemic would probably take place.

He had been struck by the work of Levaditi in regard to the grouping of what he might call the modern types of disease, such as poliomyelitis, encephalitis, vaccinia and rabies. All these were caused by filter-passing viruses. All the viruses kept well in glycerine; all were destroyed at the same temperature. All affected tissues arose from the embryonic ectodermal layer, skin, bucco-pharyngeal mucous membrane, central nervous system. Levaditi had contended that the conditions should be known as neurotrophic ectodermoses. To some extent these viruses were analagous in action to the treponema and this suggests the possibility of arsenic compounds being of value.

Dr. Sutton was interested that no mention had been made of "Urotropine," a drug which had previously been credited with much usefulness in such conditions as infantile paralysis. Another point to which he would draw attention, was the tenderness of the affected muscles in the disease. If as alleged the effects of the disease were due to vascular pressure on the cells, he was puzzled as to why tenderness should be present and for so prolonged a period. This meant probably that a true polyneuritis was present. Surely, too, according to the work of Royle and Hunter, the flaccidity pointed to some involvement of the sympathetic nervous system.

In regard to schools he did not think it right that they should be blamed for the spread of the disease; they were a kind of "Aunt Sally" for everybody to attack. He held that there was no evidence that infection spread by way of the schools. The Norwegian experience was fallacious and it was extremely rare in New South Wales to get more than one case even in a school of over a thousand during the epidemic. In any case most of the infections occurred in children before the age of three years and there was quite a distinct mortality in patients over sixty years of age. The serious side of anterior poliomyelitis was the permanency of the paralysis. One half of all the cripples owed their condition to poliomyelitis. Invalid pensions were being paid to three hundred and fifty persons in the Commonwealth on account of infantile paralysis. He had recently investigated the cripples among six thousand children. Fifty-three had been crippled in some form or other. In twenty-seven the crippling was due to infantile paralysis. Of this number twelve had lost one hundred and fifty months of school life on account of treatment. Five of the remaining twenty-six cripples suffered from tuberculous lesions of the bones and joints, in eight the condition was congenital, seven had been injured at birth and in the remainder the cause had been accidental injury.

Dr. John MacPherson referred to the use of "Urotropine" or hexamin as it was generally called. It had been previously used because it was supposed to be excreted in the cerebro-spinal fluid by the chorioid plexus. This had been disproved by some work published in *The Journal of the American Medical Association*. Even after massive doses no trace of the drug had been found in the cerebro-spinal fluid. Moreover, "Formalin" was only liberated in an acid medium and it was inconceivable that this would occur in the cerebro-spinal fluid.

Dr. J. L. T. Isbister said that Professor J. Burton Cleland, of Adelaide, had known of the discussion to be held that evening and had sent him a short statement which he proposed to read. Professor Cleland stated that the question as to whether any measures could be taken to minimize the likelihood of the introduction of the virus of infantile paralysis (acute polio-encephalo-myelitis) from New Zealand or other parts to Australia, necessitated a consideration of the epidemiology of this and of allied diseases and a review of what was known respecting the viruses of the group. Unfortunately, as would be seen, there were no practicable means by which the introduction could be reasonably well prevented and there was little prospect of the future giving more control over the transmission of the disease.

Acute polio-encephalo-myelitis belonged to a group of diseases which included "X-disease." He believed this to be a definite entity. It also included *encephalitis lethargica*. The viruses of these diseases apparently waxed and waned in virulence from time to time. Hence epidemics of each occurred. In the intervals odd cases alone were met with, as in infantile paralysis, or the disease might apparently disappear for many years, as in the case of lethargic encephalitis.

The case of infantile paralysis showed that though examples of the disease might be rare in the interepidemic interval, nevertheless the virus must be present continually to enable the few cases met with annually to occur at all. During an epidemic the number of cases increased. There was every reason to think that very large numbers of the community harboured the virus from time to time, but that it was only a very occasional individual in whom the virus was able not only to establish itself, but also to call forth the symptoms of the disease. The cases of the disease that occurred in an epidemic had often no relationship at all, direct or indirect, to previous cases. There were clear instances in which in one or other of this group of diseases healthy carriers must have conveyed the disease to isolated country districts.

This group of diseases might be looked upon as one in which the human individual was relatively insusceptible, but in which there was still a certain small proportion of persons who might become infected or succumb to the virus. This relative insusceptibility might be reduced to some extent by climatic and seasonal factors. Thus in-

fantile paralysis tended to occur in epidemic form in the autumn. It would be expected to die out or diminish towards the end of winter and the beginning of spring. The Australian X-disease occurred in the late summer and autumn and was almost confined to the hot and dry interior parts. During the epidemic of this disease some years ago in western New South Wales it had seemed certain that the virus must have been introduced into Sydney on many occasions and yet failed to establish itself there, though it had done so at once when it was conveyed outwards to some fresh focus in the interior.

The position might be summed up as follows:

(i.) The virus of infantile paralysis (acute polio-encephalo-myelitis) became very widely distributed throughout the community during an epidemic. It might be equally widely distributed, but of feeble virulence, during the intervals between epidemics.

(ii.) Only a very small proportion of children and adults who harboured the virus, became sufferers from the disease.

(iii.) The only means of ascertaining the presence of the virus in a carrier would be by monkey inoculations.

(iv.) Many or few passengers from New Zealand to Australia would, if the epidemic in New Zealand reached large proportions, be carriers of the virus. It would not be practicable to detect these carriers, as monkey inoculations would be required. Entire cessation of communication with the affected part of the world would be the only means of preventing entry.

(v.) Recovering patients and immediate contacts were more likely to harbour the virus than the population at large, but considering the evidence that pointed to the virus being very widely spread, it seemed doubtful whether any real advantage would follow isolation of such contacts or patients.

(vi.) With the rapid coming on of winter, the disease if introduced would probably not establish itself to a grave degree in 1925 in Australia, though it might recur in the autumn of the following year.

(vii.) He did not think that any process of spraying or fumigation applied to all passengers and crews of New Zealand vessels, would be likely to free carriers of the virus.

Professor Cleland concluded that there would seem, therefore, to be no reasonable means by which the likelihood of the introduction of the virus of infantile paralysis from New Zealand could be in any way lessened and doubtless there had already been quite a number of such introductions.

Dr. R. B. Wade, the President, said that he would like to make some remarks from the clinical side. In regard to early diagnosis he thought that the hyperæsthesia of the muscles was a great help. Again, in some children it was not uncommon to find retention of urine as an early symptom. As Dr. Clubbe had said, there was considerable variation in the virulence of the disease. In the 1916 epidemic in New South Wales the infection had been mild and the extent of the paralysis had not been great. Speaking purely from memory he thought that among ninety or more children treated at the Royal Alexandra Hospital for Children complete recovery of muscles had occurred in about 60% of the cases. On the other hand, it was quite impossible to give any satisfactory prognosis at all. Anything might happen. A patient might have two limbs affected, one might recover and the other not. In regard to early splinting he was not in agreement with all that had been said. He thought that if a comfortably padded splint were applied to the child in the most restful position, relief from pain would be obtained and so the child would be more comfortable. In any epidemic only 10% of the encephalitic type of the disease were seen and it was the exception in after life to see any residual effect of the upper motor neurone. Apart from the cases of hemiplegia, he had seen only two. In regard to contagion no attempt at isolation of patients had been made at the Children's Hospital during the 1903 outbreak. He had seen one case only occur *de novo* in hospital and there was nothing to show that the child had become affected in the institution; in fact, it appeared much more likely that infection had been a chance one from outside. In regard to the virulence of epidemics he referred to one which had occurred in Ballina twenty years previously. The epidemic had been a severe one, but fortunately it had been confined to the one town. Among the patients in this epidemic he had seen some examples of two infections in the one family and one example of three in a family.

At the Annual Meeting of the Western Australian Branch of the British Medical Association held at the Hospital for the Insane, Claremont, on March 22, 1925 (see The Medical Journal of Australia, May 2, 1925, page 462).

Dr. E. J. T. Thompson read notes on two cases of katatonic stupor (see page 541).

Dr. J. Bentley, M.C., read notes of a case of manic depressive psychosis (see page 542).

Both speakers called attention to the dermographia which was present in all the patients presented, notwithstanding the different characteristics of the conditions from which the patients were suffering.

Dr. D. M. McWhae, C.M.G., C.B.E., spoke of a patient in whom the glands of internal secretion were badly at fault. In both dermographia was readily elicited.

Expression of Thanks.

The President pointed out that it was through the kindness of Dr. J. T. Anderson, the Inspector-General of the Insane, that the general meetings of the Branch had been held for many years at the Claremont Hospital. Dr. Anderson had also entertained the members in a very hospitable manner. He wished to assure Dr. Anderson that the Branch and the Council appreciated his kindness and offered him the thanks of the meeting. He said that it had given the members of the Branch great pleasure to accept Dr. Anderson's invitation.

Dr. J. T. Anderson said that it was very nice to receive all the thanks, but that his colleagues had done all the work; he had merely done "the rest." He thought that it was of use for members to see patients in the mental hospital, since they had no teaching school in Perth. It had been the wish of many of the members that patients with the commonest forms of insanity should be demonstrated. They could walk around Europe and not find a finer contrast than that presented by the three patients with stupor. In conclusion Dr. Anderson expressed the hope that the meeting at the mental hospital would be regarded as an annual fixture.

Medical Politics.

NATIONAL HEALTH INSURANCE.

(*Continued from Page* 524.)

2. National Health Scheme.

As previously stated, your commissioners are of the opinion that a national health scheme should be instituted which will aim at adequate medical treatment for the people and which will provide the requisite machinery for the prevention of sickness and accident; and also that the health scheme should be dissociated from the administration of the national insurance fund. It is further recommended that the functions and objects of the Health Department be extended in such manner as will enable provision to be made as early as possible for the effective supervision of adequate medical services, especially with respect to maternity treatment.

The functions and operations of existing organizations in Australia and the questions which will require consideration at the inception of a national health scheme, are as follows:

Medical Benefits.

(a) *Medical Attendance.*—Friendly societies in Australia make arrangements wherever possible with local medical practitioners for medical attendance on members, such

arrangements being generally in the form of a contract although not always signed between the individual branch of the society and the medical practitioner, in accordance with the model form of agreement accepted by the State branch of the British Medical Association and the Friendly Societies' Association. The medical benefits are supervised by the branches of the society and the members thereof have freedom of choice with respect to the lodge doctor by whom they desire to be attended. These agreements are terminable at any time upon three months' notice by either party. The form of agreement is the result of considerable negotiations between the parties concerned and there is a variation between those entered into in the several States owing to each State organization having settled the question separately.

A member joining a friendly society since the date of the agreement is only entitled to medical attendance if his income (including, if married, that of his wife) at the time of joining is less than the income stated in the agreement. The annual incomes prescribed by the agreements in the several States are as follows: New South Wales £260; Victoria, single £260, married £312; Queensland, single £208, married £260; South Australia £312; Western Australia, single £260, married £320; Tasmania £208. The member continues to be entitled to medical attendance as long as his income does not exceed in New South Wales £364 *per annum*, in Queensland £400, in South Australia £450, in Western Australia £400 and in Tasmania £312. A special proviso is usually made with respect to members having a large number of dependants.

The lodge doctor is remunerated on the basis of an agreed contract rate per member per quarter and in accordance with the number of members on the list forwarded to him by the secretary of the branch of the society at the beginning of each quarter. The medical fee is paid irrespective of whether the member receives medical attendance or not. This remuneration covers the cost of treatment of the lodge member, his wife and dependent children up to the age of sixteen in the case of males and to age eighteen in the case of females, the widowed mother of an unmarried member, if wholly dependent, the widow of a deceased member and dependent children and the dependent brothers and sisters of an unmarried member.

The most important variation in the terms of the agreement is that relating to the contract rate of remuneration payable to the medical practitioner, which varies appreciably in each State, the annual rate per adult member payable in the metropolitan area in the several States being as follows: New South Wales 26s., Victoria 20s., Queensland 24s., South Australia 41s. (including medicine), Western Australia 24s. and Tasmania 20s.. The country rate varies considerably according to the district and is invariably higher than that for metropolitan areas, being in some districts as high as 44s. per member *per annum* for medical attendance only. In country districts a mileage fee at a rate varying from 2s. 6d. to 7s. 6d. per mile is charged in addition for travelling to the patient's home. Junior members and single female members in some instances are charged lower rates, but in others they must pay the same rates for medical attendance as an adult married wage-earner, such fees helping to pay for the cost of attendance on members' wives and dependants, the contract rate of remuneration being thus regarded from an insurance standpoint. A lodge doctor is not precluded from entering into an agreement to receive more remuneration than the minimum stated in the model form of agrement, but according to the British Medical Association's ruling he must not agree to do the work for less than that minimum. A medical practitioner may and often does enter into an agreement with more than one friendly society branch.

The services to be given by the doctor cover ordinary general practice only, special services if required being performed by the doctor at an additional fee privately arranged between the friendly society member and the doctor, in which case a reduced fee is usually charged. A list of minimum charges for such special treatment is in some States attached to the agreement. As the scope of treatment is thus limited to minor ailments only, it has been suggested that the agreement should be extended to provide for treatment of a special nature and which must now be paid for separately, in order that breadwinners may obtain the services of surgeons for major operations at reasonable rates, as the majority are unable to afford major operations and many are debarred from entering public hospitals. The national insurance scheme in England only provides for a somewhat similar medical service to that now in operation under the friendly societies system in Australia. It has been suggested that an agreement, having as its basis the payment of a flat rate per attendance, would be more satisfactory and equitable for all concerned than the present contract rate.

It is estimated that in Australia about 40% of the medical practitioners who are in practice at the present time, are carrying out friendly society work. Most doctors when commencing in a new district are available for friendly society work; most country medical practitioners undertake lodge practice and it is stated that most metropolitan doctors have at one stage in their career carried out the duties of a lodge doctor. On the other hand many medical practitioners will not undertake friendly society practice, as their services are otherwise fully engaged. No limit is placed on the number of friendly society members allotted to a doctor, as the largest branch of a society seldom exceeds five hundred members and the occasion or necessity for such limitation does not often occur, although under the present system one medical practitioner may be appointed lodge doctor for several lodges in the district. A medical practitioner cannot satisfactorily and adequately deal with more than a certain number of patients. In some areas the people are more liable to disease, consequently a standard number of patients cannot be set for all localities; in other districts a large percentage of lodge patients seldom require the doctor's services. Wives and children of members make a heavy practice and the doctor generally requires considerable time for his ordinary private practice. It is stated that five hundred members can be adequately attended by one doctor, eight hundred become a burden and one thousand are considered overwork. In some localities the lodge doctors have an arrangement whereby they work in pairs and this arrangement together with a system of centralization of patients tends to increase the number that can be attended. In country areas where there are difficulties in organizing the work and considerable travelling involved, three hundred members are considered the maximum. In the early days of national insurance in England there was no limit to the numebr of insured persons allotted to a doctor, but now as the result of experience it has been found necessary to fix a limit.

Friendly societies in some districts have associated together for the purpose of forming a medical institute, having as its object the provision of medical attendance for members, such medical attendance being carried out by a full-time medical officer employed by the institute. There has been considerable difficulty, however, in obtaining medical practitioners who will do the work under those conditions, as the British Medical Association is entirely opposed to such arrangements. The annual rate of contribution paid by members for the services of the medical institute doctor is generally less than that paid under the common form of agreement entered into with private practitioners. In a few instances a hospital has been established in connexion with medical institutes for the institutional treatment of members, it being provided that the patient must have been a member of the institute for a period of twelve months prior to treatment, in which case a nominal amount of from 15s. to £1 per week is charged to the member for his treatment and maintenance whilst in hospital.

It has not been possible to obtain complete data as to the average number of occasions on which a lodge doctor is required to attend a lodge member and his dependants during the course of a year. The particulars available, however, show a variation of from seven to fifteen visits *per annum* for the lodge member, his wife and family, representing an approximate average of about ten visits per member *per annum*, of which three would

represent visits to the patient's home and seven consultations in the doctor's surgery. Under national insurance in England the average is 3.5 visits per member *per annum* for the insured person alone and in Scotland the average is approximately four visits for the member annually, medical benefit in the United Kingdom being provided for the insured person only at a contract rate of 9s. per member *per annum*, medical benefit for a member's wife and family being obtained if required through the voluntary friendly society system.

During the year 1922 medical attendance and medicine made available to friendly society members in Australia cost £671,921. During the period from 1870 to 1915 the cost of medical benefit per friendly society member throughout Australia oscillated slightly from year to year and was on the average less than 21s. per member for medical attendance and medicine. In 1915 it was 20s. 5d. and since then there has been a continuous increase up to 26s. 5d. in 1922, representing an increase of 29% as compared with a rise of 25% in wholesale prices and 62% in wages for the same period.

Medical benefits are considered by friendly societies to be essential to their objects, as they are a great attraction to new members and the societies do not favour opening a branch where a doctor is not available. The majority, so it has been stated, join friendly societies in order to obtain medical benefits and this statement supports the opinion that the medical service is satisfactory and is now working fairly smoothly, although with a service providing in Australia for over 524,000 friendly society members and their dependants it is only to be expected that difficulties will ocassionally arise.

The present system of medical attendance arranged by friendly societies is stated to be fairly adequate for the purpose required, although the tendency in recent years, so it is suggested, has been to cut down the service included in the contract to the lowest possible limit, but there is no complaint, however, as to the standard of the service rendered. In the metropolitan areas medical benefits are more nearly adequate than in country districts, as public hospitals usually provide facilities for special treatment such as X-ray, bacteriological or vaccine treatment. If the aim to be attained is that adequate medical treatment shall be available for all requirements, the present arrangements will require considerable extension and coordination. Outside the metropolitan areas there are few doctors who practise as specialists and there are inadequate facilities locally for special examination and treatment.

At the present time there is one doctor to every 1,400 of population in Australia and it has been stated that the population of Australia is well served as regards medical attendance; on the other had although the poor are provided for by public hospitals and the rich by private hospitals, yet the middle class, wherein is included the majority of the population, is debarred the benefits available to the needy and has to provide for itself without any assistance. National health insurance, so it is stated, tends to commercialize medical practice and the medical profession is not agreeable to any existing form of national health insurance being instituted in Australia, although it has no objection to the principle of the friendly societies' medical service, but it objects to an extension of contract practice. No doctor, so it has been suggested, should be compelled to undertake contract practice, as his services would be of less value if such compulsion were used and further that under a national insurance medical service the relations between the patient and the medical practitioner are changed, generally to the detriment of the patient's interest by the intrusion of a third party in the form of a Government representative. Once a doctor agreed to do such work, however, he should be compelled to act in accordance with the regulations and be subject to a penalty for any failure to do so. It has been further suggested that the cost of providing adequate medical service in the sparsely populated areas of Australia would be very heavy. In recent years in England a gradual change is said to have taken place in the character of the medicines ordered by medical practitioners, as the result of the institution of national insurance and the establishment of the Ministry of Health and since the inception of national insurance in England the medical profession is on a better financial basis than formerly.

The provision of medical benefit under national insurance in the United Kingdom has resulted in a most involved and difficult problem of administration and it has been suggested that far more satisfactory results would be obtained in Australia if arrangements were made whereby the insured person received an amount of sick pay per week which would enable him to make his own arrangements with any medical practitioner for the services required. Alternative suggestions have been made that the Government should engage whole-time doctors on a salary basis for any medical attendance required under national health insurance or that the medical profession should be nationalized. Your commissioners are of the opinion, however, that health supervision is not necessarily a subject for insurance and medical benefit is essentially a matter for the Health Department as a part of a national health system as distinct from a national insurance scheme.

(*b*) *Supply of Medicine.*—The arrangements for the supply of medicine to members of friendly societies are delegated by the society to the individual branches. In the metropolitan and provincial urban districts most of the lodge dispensing work is undertaken by friendly society dispensaries which have been established for the purpose. In the smaller provincial towns and in the country districts, where there are no friendly society dispensaries available or where the number of members is insufficient to warrant the formation of a dispensary, the branches have entered into contracts with private pharmaceutical chemists to undertake the work for a remuneration based on a contract rate per member per quarter. In some parts of the Commonwealth somewhat similar contracts are entered into with the lodge doctor who does the dispensing work himself, but this practice is not generally approved of and is discouraged as much as possible, although in many country districts there is insufficient population to support a chemist under present conditions and it is thus impossible in those districts to separate prescribing from dispensing. In other localities where the remuneration contracted to be paid to the lodge doctor also covers the cost of medicine, the doctor sometimes enters into a private arrangement with the chemist for dispensing the prescriptions which he issues.

Friendly society dispensaries are separately constituted organizations, the capital at the establishment of the dispensary having been furnished by the branches of the friendly societies which have affiliated for the purpose, by debentures or by loans or in a few instances by loans from the State Government. In some instances dispensaries have registered under the *Shops Act* in order to be in a position to retail chemists' sundries *et cetera* to the general public as well as to members. Each dispensary is governed by an executive appointed from representatives of the various societies connected with it. All the societies in the district generally participate in a dispensary when available. Members of trade union and employees' benefit funds in some instances are admitted to membership of the dispensaries and are eligible for the benefits provided. The majority of friendly society members usually contribute to the local dispensary, although a small percentage prefer to make their own private arrangements for the supply of medicine. Each society regularly forwards to the dispensary a list, showing the names of the members who are contributing for the supply of medicine and the executive of the dispensary assesses the amount of the members' contributions required on the basis of the cost of maintaining the dispensary; in the event of the contribution proving insufficient, it is automatically increased to meet requirements. The average contribution for the supply of medicine to a member, his wife and dependants (males under age sixteen, females under age eighteen) ranges from 8s. to 12s. *per annum*, for a single member 4s. and for a juvenile member 1s. 6d. to 2s.. In industrial areas where there is a heavy demand for medicine, a larger annual subscription is required from members. In cases where the dispensaries make a profit from the sale of chemists' sundries, this profit is used

in the reduction of the quarterly contribution required from members. For this contribution anything within the "British Pharmacopœia," when prescribed by a qualified medical practitioner, is issued by the dispensary, but patent or proprietary medicines must be paid for in addition, usually at a reduced price. Where special medicines form a part of a prescription they are generally dispensed without additional charges; certain sundries, if included in a prescription, are also supplied without charge. Some dispensaries lend certain surgical appliances to their members, usually at a small fee, but special surgical appliances must be purchased by the member at his own cost. Most dispensaries have been able to accumulate sufficient funds to repay the original loans obtained at their establishment and also to pay for the cost of the buildings occupied. The arrangements for the supply of medicines by dispensaries to friendly society members are generally held to be adequate for the purpose required, although it has been suggested there should be an extension of such benefits which would enable all drugs to be supplied under the same conditions as now operate in respect to those drugs included in the "British Pharmacopœia."

The contracts entered into with private pharmaceutical chemists vary throughout the Commonwealth, the contract rate per member ranging from 8s. to 15s. *per annum* and for this remuneration the chemist undertakes to supply medicine for the member, his wife and dependent children, males under age sixteen and females under age eighteen, in accordance with the prescriptions issued by the lodge doctor. The member has the right of selecting on which available chemist's list he requires to be placed. Special prescriptions must be paid for in addition and similar provisions apply to patent medicines, but in such cases a reduction in current prices is usually made. In some districts the chemist instead of accepting the usual *per capita* rate payment, undertakes to dispense all medicines for friendly society members at a fixed percentage, usually from 25% to 33% below current retail prices. The services rendered by private pharmaceutical chemists under these contracts are generally stated to be satisfactory, but the chemists, however, consider lodge work to be somewhat uncertain, as they are unable to estimate whether the contract rate will be sufficient to cover the expenditure during any period. The charges made by dispensaries for the supply of medicine to members are generally lower than those of private chemists for similar work, as there is a greater volume and continuity of dispensing work in a dispensary which is also not subjected to the same overhead expenses as a private chemist and also owing to the fact that lodge prescribing is usually based on a standard pharmacopœia and this system enables a large percentage of stock medicines to be used.

Full particulars, showing the average number of prescriptions dispensed per friendly society member *per annum* cannot be readily obtained from private pharmaceutical chemists undertaking lodge work, but considerable data have been made available by the various united friendly society dispensaries operating in the several States of the Commonwealth. Particulars were furnished from thirty-eight dispensaries in the Commonwealth, showing that in the year 1923 a total of 1,918,484 prescriptions were dispensed for 231,039 members, representing an average of 8.3 prescriptions dispensed *per annum* in respect of the member, his wife and dependants. The estimates supplied by private chemists indicated a range of from six to fifteeen prescriptions per member *per annum*. It is estimated that from 50% to 60% of the prescriptions were mixtures, 5% to 9% ointments, 5% to 8% lotions, 5% to 10% liniments, 5% to 7% powders, 3% to 4% pills and the balance consisted of special preparations. Estimates which have been made by dispensary officials, show that about 35% of the prescriptions were repeated. Repeat prescriptions were abolished under national insurance in England in 1920, owing to the danger of a repeat prescription being issued to other than the insured member and also on account of the time occupied by the chemist in looking up the original prescription record. The above prescriptions dispensed by the friendly society dispensaries averaged a total cost of 12.6d. per prescription, representing 5.3d. for drug contents and 7.3d. for the cost of dispensing and overhead charges, although owing to the fact that profit is made on cash sales it is not always possible to accurately arrive at the actual cost per prescription. In private pharmacists' business this is especially so and particulars were furnished showing that the cost of lodge prescriptions ranged from 15d. upwards. It has been stated that if chemists were fully occupied on dispensing work and the doctors wrote prescriptions in accordance with a uniform pharmacopœia, the dispensing work might be done at a total average cost of 1s. per prescription, the cost of dispensing in friendly society dispensaries being similarly reduced as the result of a larger membership.

Under the national health insurance scheme in England the cost per insured person for medicine only is about 2s. 7d. *per annum*, the cost of ingredients per prescription being approximately 4d., to which a dispensing fee of from 5d. to 6d. per prescription is added, the whole of the dispensing work under the *National Insurance Act* being carried out by private chemists. If the national insurance system in the United Kingdom had provided medical benefits for the family of the insured person, such would, so it is suggested, have had a serious effect on the voluntary side of friendly societies' efforts, because the most important factor in friendly society work is stated to be the desire of the breadwinner to have his wife and children included under the medical benefits provided by the friendly societies' medical service.

Under the present contract system a definite rate is paid per quarter for each member, irrespective of whether the member receives medicine or not, with the result that the remuneration is higher per prescription when members are healthy and when fewer prescriptions are dispensed than at the time of an epidemic when more prescriptions are dispensed. The average cost of prescriptions varies in certain districts, owing to some medical practitioners prescribing more expensive ingredients than others. The most desirable system for payment of medicines is stated to be that having for its basis payment for services rendered at an average flat rate per prescription dispensed, a uniform pharmacopœia being adopted for the purpose and the cost of special prescriptions being met from a special fund earmarked for the purpose. A uniform pharmacopœia would be essential in order that the scheme could be run on economic lines, as it would be a means towards quick work and time saving and in order that a basis for the calculation of a prescription flat rate could be effectively ascertained.

(c) *Other Medical Benefits.*—No general arrangements for dental treatment have been adopted by friendly societies, although in some branches arrangements have been made for the provision of dental benefits for members at reduced rates. Provision is made by some societies whereby, for the payment of a contribution of 2s. 6d. *per annum*, a payment of from 15s. to 20s. per week is made in respect of a member undergoing treatment in any hospital in the Comomnwealth.

(To be continued.)

Correspondence.

VENEREAL DISEASES.

SIR: I believe the *Venereal Diseases Act, 1918* (New South Wales) to have failed.

As it stands it is a menace. It does what it was not intended to do—drives infected persons into the hands of "quacks." I include chemists who treat venereal disease, under the term "quack."

Since the Act was introduced I have watched its effect with some interest and have noted some of its weak points. I know that venereal disease is very common in Sydney and I doubt if even half the infected persons are being treated by legally qualified medical practitioners. Very many persons are being treated by chemists who give them unlabelled and unmarked bottles or who post their medicines to them instead of handing them over the counter.

There are various laymen in Sydney and suburbs who treat venereal diseases. All these people do harm. They profit and the community pays. Their treatments are mostly futile and even though the patient may show no external signs of disease, heaven alone knows what menace lies latent in his body.

The greatest mistake in the *Venereal Diseases Act* lies in the section which deals with notification. The majority of infected persons will not go where there is a possibility of their name becoming public and so they go to a man who for his own protection does not notify them.

Why not repeal the Act and start afresh on common sense lines? At present we are only wasting time and money and are doing more harm than good.

I do not know who was responsible for the Act in its present form, but it gives one the impression that its sponsors had but little knowledge of the mental outlook of the venereal patient.

Yours, etc.,

J. COOPER BOOTH.

171, Macquarie Street,
April 17, 1923.

Books Received.

ENCYCLOPÆDIA MEDICA, under the General Editorship of the late J. W. Ballantyne, M.D., C.M., F.R.C.P.E. (Volumes I. to VIII.) and Alexander Goodall, M.D., F.R.C.P.E. (Volumes IX. to XIII.); Second Edition. Volume XIII.; Tularæm to Zinc Supplement; 1925. Edinburgh: W. Green & Son, Limited; Sydney: Butterworth & Company (Australia), Limited. Royal 8vo., pp. vii. + 671, with illustrations.

Medical Appointments.

Dr. V. R. Delany (B.M.A.) has been appointed Officer of Health to the East Torrens Local Board of Health, South Australia.

Dr. Clarence Oscar F. Reiger (B.M.A.) has been appointed Jusitce of the Peace for Zeehan, Tasmania.

Dr. Constance Alice Finlayson (B.M.A.) has been appointed Honorary Clinical Assistant in Biochemistry at the Adelaide Hospital.

Dr. Leslie James Keipert (B.M.A.) has been appointed Public Vaccinator at Rupanyup, Victoria.

Dr. Francis Florence D'Arcy (B.M.A.) has been appointed Public Vaccinator at South Yarra, Victoria.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi..

ADELAIDE HOSPITAL: Bio-Chemist.
FEDERAL CAPITAL COMMISSION, CANBERRA: Health Officer.
MARRICKVILLE DISTRICT HOSPITAL, SYDNEY: Honorary Consultant Surgeon for Ear, Nose and Throat Department.
MELBOURNE HOSPITAL: Medical Vacancies.
PUBLIC SERVICE BOARD, NEW SOUTH WALES: Medical Officer (Female).
PUBLIC SERVICE COMMISSIONER, VICTORIA: Medical Inspector of Factories and Shops.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY: Honorary Assistant Ophthalmic Surgeon.
ROYAL AUSTRALIAN AIR FORCE: Medical Officer.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, SYDNEY: Resident Medical Officer.
ST. VINCENT'S HOSPITAL, SYDNEY: Honorary Physician to In-Patients and (2) Honorary Physicians to Out-Patients.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

MAY 26.—New South Wales Branch, B.M.A.: Medical Politics Committee: Organization and Science Committee.
MAY 27.—Victorian Branch, B.M.A.: Council.
MAY 28.—New South Wales Branch, B.M.A.: Branch.
MAY 28.—South Australian Branch, B.M.A.: Branch.
MAY 29.—South Eastern Medical Association, New South Wales.
JUNE 2.—Tasmanian Branch, B.M.A.: Council.
JUNE 3.—Victorian Branch, B.M.A.: Branch.
JUNE 3.—Section of Obstetrics and Gynæcology, New South Wales Branch, B.M.A..
JUNE 4.—Section of Orthopædics, New South Wales Branch, B.M.A..
JUNE 5.—Queensland Branch, B.M.A.: Branch.
JUNE 8.—Northern District Medical Association, New South Wales.
JUNE 9.—Tasmanian Branch, B.M.A.: Branch.
JUNE 9.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 11.—Victorian Branch, B.M.A.: Council.
JUNE 11.—South Australian Branch, B.M.A.: Council.
JUNE 11.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 12.—Western Australian Branch, B.M.A.: Council.
JUNE 12.—Queensland Branch, B.M.A.: Council.
JUNE 16.—Tasmanian Branch, B.M.A.: Council.
JUNE 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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